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# ONE HUNDRED UNTREATED CANCERS OF THE RECTUM*

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THE success of the treatment of any disease is measured by a study of the cases treated in comparison with a group which is untreated. It is essential to study the natural history of cancer to determine the benefits of treatment. Although the importance of such a study seems obvious, it is surprising that very little such information has been presented in the literature.

The only adequate series of untreated cases of cancer of the common types in other countries are presented by the British Ministry of Health. Large series of cancers of the breast, uterus, rectum, tongue and mouth, and esophagus have been studied by this committee[1, 2, 3]. Untreated cancer of the breast has been studied in Switzerland by Lukac[4]. In this country a start has been made by a survey of one hundred untreated cancers of the breast by Daland[5], but other groups of cancer have not been analyzed.

Cancer of the rectum is one of the most common types of malignant disease. We have chosen it for this discussion because of the comparatively large number of untreated cases that we have been able to observe, and because we have also been able to see the results of treatment by other methods in the same hospitals.

TABLE 1

DISTRIBUTION OF UNTREATED CASES

| Hospital | No. Cases | Years |
|---|---|---|
| Collis P. Huntington Memorial | 44 | 1912-35 |
| Pondville Hospital | 41 | 1927-35 |
| House of the Good Samaritan | 10 | 1912-32 |
| Massachusetts General Hospital | 5 | 1910-32 |
| Total | 100 | |

These one hundred cases have been taken from the Collis P. Huntington Memorial Hospital, The Pondville Hospital, The House of the Good Samaritan and the Massachusetts General Hospital. The three hospitals first mentioned are cancer hospitals and all cases in their record files have been considered. The Massachusetts General Hospital is a hospital for acute illnesses, but we were able to find five who died without treatment. All records have been checked very carefully; letters have been written to hospitals, physicians and relatives; we have reviewed the death certificates and feel certain that none of the patients mentioned in this series ever received any treatment. A microscopic diagnosis of carcinoma was made in thirty-five cases, either from biopsy or postmortem material.

The length of life, the comfort of the patient and the comfort of his family are the only measures of comparison between treatment and nontreatment. The length of life can be accurately determined and furnishes a valuable index of the benefits of various types of treatment. The comfort of the patient, on the other hand, cannot be reduced to figures, but there is hardly a more miserable man alive than one with an advanced cancer of the rectum. This is so well realized that many surgeons feel that desperate chances should be taken to attempt a cure by radical surgery rather than let the patient go on to the advanced stages. Not only is the untreated patient ever in distress, but he is a source of trouble to his whole family. A well-functioning colostomy opening is much easier to care for than an incontinent rectum.

All of our data have been computed from the onset of the symptoms which we interpret as due to the disease. A change in bowel habits is the criterion we have used in determining the date of onset of symptoms. The actual beginning of the cancer must have antedated the first symptom.

Why were these patients untreated? We cannot give an exact answer. Some patients were too old or in too poor condition. Some were deterred by the thoughts of a colostomy or by the magnitude of the radical operation. The disease in some patients when first examined was too advanced for treatment. In still others, the medical advice before admission to the hospital was poor, because of inadequate rectal examina-

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tion, prejudice against colostomy or lack of faith in surgery.

There were fifty-six males and forty-four females in our series. The youngest was thirty-two and the oldest ninety-two. The average age at onset of symptoms was 59.6 years. The median age was fifty-nine years,—that is, there were as many under fifty-nine years as there were over it. Table 2 shows the age distribution by five-year periods. The cases are fairly evenly distributed between the ages of forty-five and seventy-five. Twenty per cent of the cases were found outside of these limits. Table 2 also shows the duration of life from onset of symptoms to death in each age group. There is no significant variation in the length of life in the various groups. The average length of life was 17.2 months. The median length of life was fourteen months. One patient died one month after onset. The longest duration was in one patient who lived forty-nine months. The females lived slightly longer than the males; the median length of life was sixteen and fourteen months, respectively. The accumulated death curve of all untreated cases is shown in figure 1.

TABLE 2

AGES AND DURATION OF LIFE FROM ONSET OF SYMPTOMS

| Age | Untreated Cancer of Rectum | | Colostomy Only | | Colostomy and X-Ray Treatment | |
|---|---|---|---|---|---|---|
| | No. Cases | Av. Duration Life | No. Cases | Av. Duration Life | No. Cases | Av. Duration Life |
| 30-34 | 1 | 25 (months) | 3 | 19 (months) | — | 4 (months) |
| 35-39 | 3 | 12.3 | 0 | — | 1 | 22.7 |
| 40-44 | 6 | 23.8 | 9 | 11 | 4 | 17.5 |
| 45-49 | 11 | 18.5 | 8 | 19 | 7 | 33 |
| 50-54 | 14 | 18.6 | 14 | 19 | 3 | 32 |
| 55-59 | 17 | 14.9 | 12 | 19 | 2 | 16.5 |
| 60-64 | 12 | 25 | 14 | 12 | 4 | 11.5 |
| 65-69 | 11 | 18.2 | 10 | 20 | 6 | 12.5 |
| 70-74 | 15 | 15.2 | 6 | 16 | 4 | 24 |
| 75-79 | 7 | 11.4 | 3 | 23 | 1 | |
| 80-84 | 1 | 15 | 0 | — | | |
| 85-89 | 1 | 19 | 1 | 14 | | |
| 90-94 | 1 | 23 | | | | |
| | 100 | All died of Cancer | 80 | All died of Cancer | 32 | All died of Cancer |
| | Median length of life from onset—14 months. Average length of life from onset—17.2 months. | | Median length of life from onset—14 months. Average length of life from onset—16.9 months. | | Median length of life from onset—15 months. Average length of life from onset—18.8 months. | |

COLOSTOMY

In order to determine whether performing colostomy prolongs life, we have analyzed the records of eighty patients in whom colostomy was done without any other treatment. Sixty-five of these were from the Pondville Hospital and fifteen from the Collis P. Huntington Memorial Hospital. Fifty-nine were males and twenty-one were females. The average age at onset was 58.1 years, the median age was fifty-eight. All of these patients died of cancer, the length of life varying from two to fifty-six months after the onset of symptoms. One quarter of these patients were dead in seven months,

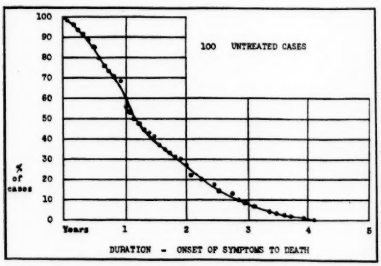


FIGURE 1. Duration of life in 100 untreated cancers of the rectum.

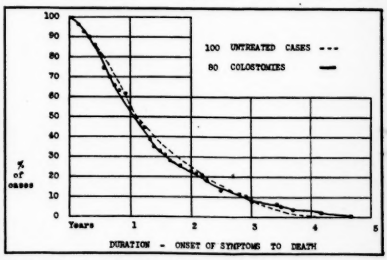


FIGURE 2. Duration of life in 80 colostomies compared with 100 untreated cancers of the rectum.

one half in fourteen months, three quarters in twenty-four months after onset of symptoms. The median length of life from onset to treatment was seven months, from treatment to death, five months and from onset to death, fourteen months. The average length of life following colostomy was six months, the average from onset to death was 16.9 months.

Table 2 shows the average duration of life in the different age periods. The youngest patient was thirty-two and the oldest eighty-eight. It will be observed from the table that the age of the patient bears no relation to the duration of the disease. Figure 2 shows that patients who have had colostomy done live no longer than the untreated cases, in fact the two curves of duration of life are practically identical. The comfort of the patients then is the only consideration in performing a colostomy.

We have included in this series twenty-four patients who have come to us after having had a colostomy done elsewhere. Of the fifty-six others in whom we advised colostomy there were seven strictly operative deaths, a mortality of 12.5 per cent. Some of these operations were done in local hospitals, but the majority were done at the Pondville Hospital or the Huntington Hospital.

### COLOSTOMY AND X-RAY

The effect of x-ray treatment to the local lesion in connection with colostomy has been studied in another group of thirty-two cases. All but three of these patients were treated at Pondville. The amount of x-ray therapy in these cases varied from 300 r. to 2800 r. units. In the majority of cases the intention was to relieve pain in the pelvis. In very few was the dosage sufficient to expect any change in the tumor. It is not fair to conclude from this small series of partially treated cases that x-ray therapy is of no value. However, in these clinics we have not been impressed with its value in any case except for the relief of pain. It is of some benefit for this purpose in instances.

This series is closely comparable with the colostomy group. These were twenty-three males and nine females. The average at onset was fifty-seven years, the median age the same. All of these patients died of cancer, half of them being dead at fifteen months after onset of symptoms. The length of life from onset varied from four months to forty-five months. The median length of life from onset to treatment was seven months, from treatment to death, eight months and from onset to death, fifteen months. It is possible that the increased median length of life from treatment to death, compared with patients in whom colostomy only was done, was due to the fact that x-ray therapy was used in the patients in whom the expectation of life seemed longer. Also this group represents only those who survived the colostomy operation, for the operation was always done before x-ray treatment was given. The average length of life following colostomy and x-ray treatment was 9.5 months, the average from onset to death 18.8 months.

Table 2 shows the distribution by age groups and the respective length of life. The numbers in each group are too small to be significant statistically. The youngest patient was thirty-eight and the oldest seventy-six. Figure 3

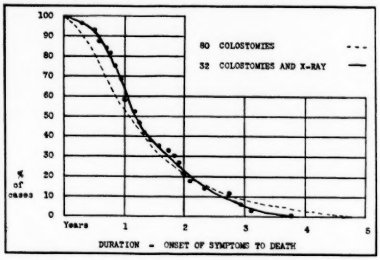


FIGURE 3. Duration of life in 32 patients having colostomy and x-ray treatment, compared with those having colostomy alone.

shows a close similarity in the curves of the patients treated by colostomy and x-ray and those in whom only a colostomy was done.

### RADICAL OPERATION

The group of forty-two radical operations here studied represents all the patients who came to the Huntington Hospital between 1912 and 1930 who were referred elsewhere for resection. None of the operations were done at the Huntington Hospital as the hospital is not equipped to do major abdominal surgery. The operations were done in nineteen different hospitals, ten of which are in Boston. End results were obtained in all cases.

There were twenty-five males and seventeen females. The youngest patient was thirty-six and the oldest was seventy-four. The average age at onset of symptoms was 54.5 years, the median age was fifty-six years. The median length of life from onset to treatment was eight months. The median length of life from treatment to death was twenty-seven months. Table 3 summarizes this series by age groups. Of the forty-two patients in whom the radical operation was done, eleven died as the result of the operation. This gives an operative mortality in the nineteen hospitals of 26.2 per cent. Thirty-three per cent were alive *five years after onset of symptoms.* Two patients died of intercurrent disease without recurrence within the five-year period. This gives us forty cases on which to base our percentage of cures. Twelve patients (30 per cent) were alive and free from disease for *five years after operation.* Seventeen patients died with cancer.

Figure 4 shows the death curve of the radical resections in comparison with that in the untreated cases. Figure 5 shows the length of life after operation in patients who had colostomies performed and those who had radical resections done.

TABLE 3

AGES OF RADICAL OPERATION CASES AND RESULTS

| Age | No. Cases | Operative Fatalities | 5 Year Cures | Died of Intercurrent Disease Within 5 Years | Died of Cancer |
|---|---|---|---|---|---|
| 35-39 | 1 | 0 | 0 | 0 | 1 |
| 40-44 | 5 | 2 | 1 | 0 | 2 |
| 45-49 | 7 | 1 | 4 | 0 | 2 |
| 50-54 | 7 | 3 | 0 | 0 | 4 |
| 55-59 | 7 | 3 | 1 | 0 | 3 |
| 60-64 | 8 | 1 | 2 | 1 | 4 |
| 65-69 | 5 | 0 | 4 | 0 | 1 |
| 70-74 | 2 | 1 | 0 | 1 | 0 |
| | 42 | 11 (26.2%) | 12 (30%)*† | 2 | 17 |

*Based on 40 cases, omitting deaths from intercurrent disease.
†Excluding operative deaths, 5 year cures are 41 per cent.

SEX

In the four groups studied above there were 254 cases. Three of the hospitals from which this material was obtained admit both men and women. The House of the Good Samaritan admits women only. Hence, in studying sex incidence, we must eliminate the ten cases from

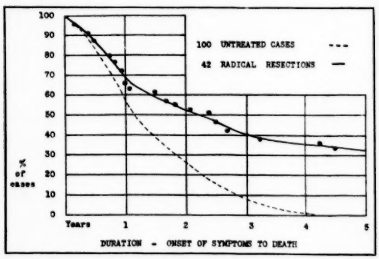


FIGURE 4. Duration of life in 42 radical resection cases compared with the untreated group.

that hospital. Of 244 cases, 163 were males and 81 were females,—exactly a 2:1 ratio.

It is generally accepted that cancer of the rectum is more common in males than in females. David[6] says that rectal cancer involves the two sexes almost equally but there is a slight preponderance in the male. In Gant's[7] series fifty-two per cent were males and forty-eight per cent females. Pack and LeFevre[8] found 69.7 per cent were females. Miles[9] quotes a 6:5 ratio between males and females. In the group where colostomy alone was possible, Gabriel[10] found a 3:1 ratio for males; Rankin, Bargen and Buie[11] found about the same ratio.

The British Ministry of Health[3] states that the operability in four published series was much higher for women than for men, and Rankin, Bargen and Buie agree that this is so.

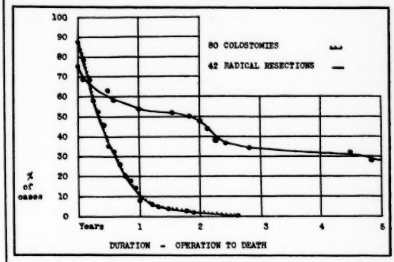


FIGURE 5. Duration of life after operation in colostomy and radical resection cases.

This may explain the reason why the female ratio is so low in the cases submitted to colostomy in the series mentioned above.

In our own study we have found that in all groups, within the limits of error, the males hold almost exactly the same 2:1 ratio, whether the patients are treated or not. This is shown in table 4.

TABLE 4

SEX INCIDENCE

| | Male | % | Female | % | Total |
|---|---|---|---|---|---|
| Untreated rectal cancer | 56 | 62.2 | 34 | 37.7 | 90 |
| Colostomy | 59 | 73.7 | 21 | 26.2 | 80 |
| Colostomy and x-ray | 23 | 71.8 | 9 | 28.1 | 32 |
| Radical resection | 25 | 59.5 | 17 | 40.4 | 42 |
| Total | 163 | | 81 | | 244 |

DISCUSSION

Reports of the British Ministry of Health show some variation in the different studies that have been made of untreated cancer of the rectum. In Report No. 33[1], 887 cases are analyzed with a median duration of life of twenty-one months from onset of symptoms. In a later report (Report No. 66)[3], comprising ninety-five cases, the median duration was 8.5 months. We are unable to explain the wide difference in their two series. Our median length of life was fourteen months, about midway between the two British series.

We agree with the following statement made by the British Ministry of Health:

"It will be seen that, either there is no significant relation between age at onset and duration or, at the least, these data are not numerous

enough to establish it. It should be remarked that the mean duration is so small in comparison with the average duration of life from any age within the range (the average after lifetime at the age of seventy exceeds 8½ years in both sexes) that the decrease of normal expectation of life with age is unimportant in comparison with the difference between the expectation of the untreated patient and that of a normal person at any age within the range."

Hayden and Shedden[12] have analyzed all the cases treated by radium at the Collis P. Huntington Hospital. They concluded that patients treated by radium alone lived no longer than if untreated, that the patients in whom colostomy has been performed lived four months longer, on the average, than if untreated and that the patients treated by colostomy plus radium lived four months longer than if colostomy they were untreated, treated by colostomy, or by radical resection.

4. The median age of the patients subjected to radical operation was three years less than in the untreated group.

5. The median delay from onset to treatment was eight months in the radical cases and seven months in the colostomy cases.

6. In this series the operative mortality rate for colostomy was 12.5 per cent and for radical resection, 26.2 per cent. The five-year curability was 30 per cent. We consider these results a fair cross-section of the results being obtained by surgeons in this community during this period, but they are by no means the optimum results that may be obtained.

## CONCLUSIONS

Patients suffering from cancer of the rectum will live as long if no treatment is given as they will if a colostomy is performed and nothing further done. However, they are much more comfortable during the fourteen months they have to live if they do submit to colostomy. X-ray treatment combined with the colostomy operation may aid in relieving pain.

The operative mortality for the radical resection of the rectum in one or two stages is low enough to warrant radical surgery when there is probability that the growth can be removed. The duration of life is much longer than in the other groups and the patients are free from symptoms during this increased span of life. There is a thirty per cent possibility of cure for at least five years.

TABLE 5

| | Untreated | Colostomy | Colostomy and X-Ray | Radical |
|---|---|---|---|---|
| Number | 100 | 80 | 32 | 42 |
| Sex | M. 56 F. 44 | M. 59 F. 21 | M. 23 F. 9 | M. 25 F. 17 |
| Average Age at Onset | 59.6 yrs. | 58.1 yrs. | 57 yrs. | 54.5 yrs. |
| Median Age at Onset | 59 yrs. | 58 yrs. | 57 yrs. | 56 yrs. |
| Average Length Life Onset to Death | 17.2 mos. | 16.8 mos. | 18.8 mos. | — |
| Median Length Life Onset to Death | 14 mos. | 14+ mos. | 15+ mos. | 27 mos. |
| Average Length Life Onset to Treatment | — | 10.8 mos. | 9.3 mos. | 8.7 mos. |
| Median Length Life Onset to Treatment | — | 7 mos. | 7 mos. | 8 mos. |
| Average Length Life Treatment to Death | — | 6 mos. | 9.5 mos. | — |
| Median Length Life Treatment to Death | — | 5 mos. | 8 mos. | 22 mos. |
| Operative Mortality | — | 12.5% (7 of 56 cases) | — | 26.2% |
| 5 Year Cures | — | — | — | 30% |

alone had been done. Their group of cases in which colostomy alone was used was small, hence, we believe, the difference between their figures and ours.

The operation for cancer of the rectum done in this series was, for the most part, either the two-stage operation advocated by Jones or the one-stage operation recommended by Jones and Miles. We cannot compare our results with those published by Jones[13] in regard to operative mortality or curability because we do not know the percentage of operability in our group.

## SUMMARY

1. The median duration of life in 100 untreated cases of cancer of the rectum was fourteen months. Untreated patients lived from one to forty-nine months after onset of symptoms.

2. Colostomy (80 cases) or colostomy combined with x-ray treatment (32 cases) did not prolong life appreciably in comparison with the life duration of untreated cases.

3. There was a two to one predominance of males over females in the cases studied, whether

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## DISCUSSION

DR. CLAUDE WELCH: Since Dr. Jarvis has already spoken of propagandizing the medical profession, you may excuse a younger man if he uses the same terms in speaking of cancer of the rectum. The value of Dr. Daland's paper, it seems to me, lies in the fact that it points out very concretely the value of radical operation in any case of cancer of the rectum.

In speaking of that fact, it would have been of great advantage if we had been able to present a curve of radical operations in which there were no operative mortalities and one hundred per cent cures. We feel that our figures do not represent the optimum, by any manner of means. For example, Dr. Jones' figures of eleven per cent mortality in his private patients and Dr. Lahey's results which were reported here last year of slightly over eight per cent in his private patients, make one realize that we have not presented these data as best we could to tell the medical profession that operation is desirable.

In order to see whether there has been any improvement in operating on these patients, I have reviewed the cases in the Massachusetts General Hospital from the year 1930 to 1934 inclusive. During this five-year period there was an operability rate of sixty-three per cent. That is a figure we could not include in our paper because we could not obtain it from the Huntington Hospital records.

This means that sixty-three per cent of all patients admitted to the wards of the Massachusetts General Hospital or Tumor Clinic were subjected to radical operation before they left. There were, during this five-year period, one hundred and seven radical operations done by twenty-two different operators, all the way from Dr. Daniel Jones down to the surgical resident.

The operative mortality in this large group was 14.1 per cent over this period. During the last year, 1934, there were twenty-nine operations done, with one death. The operability rate this year was seventy per cent. These results contrast very favorably with the slightly over twenty-six per cent mortality that Dr. Daland has reported in his cases. I think as soon as the medical profession realizes that the mortality from operation is not so high as it has been led to think previously, and that the number of five-year cures have increased remarkably, we shall gain in the treatment and control of cancer of the rectum.

PRESIDENT JOHNSON: Discussion of this paper will be opened by Dr. Lyman Allen.

DR. LYMAN ALLEN, Burlington, Vt.: I have not very much to add to this discussion. I looked up the number of cases of cancer of the rectum we had had in our two hospitals in Burlington in the last five years, but I did not receive notice that I was to discuss this in time to follow them up fully. The follow-up part of it which I tried to do was very unsatisfactory, which leads me to think that most of them have already passed beyond where we can succeed in following them up.

I found, however, that in five years in these two hospitals, of about two hundred and fifty beds, we have had only thirty-four cases of cancer of the rectum. Ten of these had no operation, whatever, and of the ten, five died while they were in the hospital, which means they were admitted in the last stages probably, too late to do anything. Twelve of the thirty-four had colostomies and we had no hospital deaths in that number.

With thirty-two out of thirty-four with either no operation or colostomy only, it means either that the cases were seen too late to make radical operation worth while, or that the patient refused radical operation. It is probably partly one and partly the other.

Five had treatment by radium and one of these cases later had a colostomy. There was no death in that list of five cases, again, evidently late cases. Only seven had radical operation and of these seven, three died, forty-two per cent. One was a cautery operation, one was a perineal operation, and five were two-stage operations. The deaths all came in the two-stage operations.

One of those radium cases, the only one I could follow, lived three years and a half, at least. The colostomies lived for varying times fifteen months, to two years. As I have said, many of them I could not follow. Our youngest patient was thirty-two and our oldest was seventy-nine.

So cancer of the rectum is not necessarily a disease of even middle age. There were twenty-two males and twelve females, which is practically the proportion given here in this paper.

It has been suggested that a complete rectal examination should be made on all the population of the country. Obviously, it is impossible to do that, but the suggestion was made in a recent paper. I do think we might say that a rectal examination should be made and could be made upon every patient that comes to us for a full check-up, as they call it. I do not think that is asking too much.

If that were done, we should get a larger percentage of early cases. As it is, since there are no early symptoms, the patient has had cancer of the rectum for some time before he presents himself to us.

In my opinion, radical operation is indicated whenever the patient will allow it. The mortality is high in the ordinary operator's hands, but I think if I had cancer of the rectum, I should welcome a high mortality.

PRESIDENT JOHNSON: Dr. Walter Seelye.

DR. WALTER C. SEELYE, Worcester, Mass.: I am very much interested in the mere fact that Dr. Daland has been able to assemble one hundred cases of untreated cancer of the rectum. I think it is a remarkable thing to have followed up so many cases and have statistics as a criterion upon which to base operative treatment.

As has been said here and emphasized so much, the mortality of cancer of the rectum by radical operation is high, but the tables which have been shown are very interesting in that they bring out the fact that even with the high mortality, high operative mortality, you have a five-year cure of thirty per cent in Dr. Daland's series, and thirty-four per cent, I believe, in Dr. Jones' series of five-year cures.

All that goes to show that in the first place technic can be improved to reduce the operative mortality, and in the second place, the cases of five-year

cure can be markedly increased if the length of time between beginning of symptoms and operation is reduced.

There is one thing that has impressed me very much in going over this subject, as Dr. Daland has presented it, and as had been presented in other papers of Dr. Jones. They emphasize an average of eight months' duration, and it is interesting to see that all statistics agree so closely in just about the eight months' period from onset of symptoms to the time the patient is brought in for treatment.

The only way I can see to shorten this interval of eight months is by a propaganda of education, first, to physicians in general, to recognize early symptoms of cancer of the rectum, and, secondly, the same education carried across to the general public to become mindful of the possibility of cancer of the rectum when they have symptoms of bleeding or mucous discharge from the rectum or change of bowel habits for which they seek advice.

That, I feel, also could well be taken up by the medical societies by sending out leaflets or reprints to all the men of each district society, to give them warning in their general examination of patients to be mindful of the possibility of cancer of the rectum in dealing with intestinal symptoms of the patients who consult them.

DR. EDWARD L. YOUNG, JR., Boston, Mass.: There are two curves Dr. Daland did not give us. One I think he never can give us, and one I wish he would. I wish some day he would give us a curve of the difference between the cures in those cases of radical operation where there was no evidence of spread of the disease and in those cases where there was a spread of the disease, and, nevertheless, the local growth was removable.

I think all of you who have seen the end stages of cancer in the pelvis, whether it comes from rectum, cervix, or bladder will feel as I do that it is a terrible way to die. I believe that cancer of the rectum should always be removed, regardless of whether there is a liver metastasis, provided the local growth is removable, because the second curve, which he can never give, is the difference, even though length of life was not prolonged, between the comfort in dying without the growth and with it.

DR. FRANK H. LAHEY, Boston, Mass.: I dislike to repeat previous statements relating to cancer, but we have definite convictions on this subject. I think that everyone who has had experience with cancer of the rectum, because of the depressing reaction to it on the part of patients, is duty bound to speak on the hopefulness of the subject. I wish to add my words to what Dr. Welch has said, that it is a hopeful lesion from the point of view of curability, also that the operability can be much increased.

Lyman Allen, sitting beside me, asked me what our mortality was. When our operability was around fifty-four per cent, our mortality was eight and a quarter per cent, but we have increased operability to seventy per cent, with the result the mortality has gone to about 12¼ per cent. We must not, as Dr. Young said, be too proud of our mortality rate because, we must widen this operability rather to make these people comfortable than narrow it to make our result appear well. If a radical removal is done and they die of liver metastasis, they are infinitely more comfortable than with local growths.

Carcinoma of the rectum is a relatively benign growth. Forty-six per cent of all our cases that have had the radical operation are alive and well over five years. When you have a lesion that can give such high percentage of relief over five years, then I believe it is your duty to get this fact before the public and dispel this depressed attitude which is in the mind of the public regarding the hopelessness of cancer of the rectum.

DR. EDWARD H. RISLEY, Waterville, Me.: I would like to say something about the treatment of colostomies. I think we all know that it is very difficult to get some patients to consent to colostomy. They dread the thing. They have heard about the difficulties in its care, and it is often very hard to persuade them to consent to it. Therefore, any detail we can add in the aftercare of our colostomy patients is very much worth while.

For the last two or three years we have been doing all our colostomies in the midline, regardless of whether they are inoperable cases for two reasons: One, because we thought it shortened our period of operation somewhat, and because it is so much more convenient for the patient to handle a colostomy in the midline.

The detail that we have worked out which is being much appreciated is very simple. The ordinary textbook illustration is of a patient handling an extensive colostomy, an irrigating can with an irrigating tube, the patient sitting on a stool with a large sheet of heavy rubber wrapped around him draped into a pail.

That is very abhorrent to most people. We have found lately that if we provide our patient, before he goes out of the hospital, with a small stiff rubber apron, which ties around the waist with a rubber strap, and is more or less pear-shaped, it drops down into the toilet. The patient sits on the toilet in the normal position.

The colostomy belt is simply strapped around the waist below the colostomy, or if the colostomy is very protruding, a little hole is made in the rubber, and it fits tight to the body over the colostomy. A little button can be put on so that it forms a cone. The patient can have his normal stool without soiling his person, and if he has to irrigate, also without soiling his person or the toilet.

This is a simple thing, but I have had a number of patients who expressed their appreciation of this little gift that I always present to them before they leave the hospital.

DR. LELAND S. MCKITTRICK, Boston, Mass.: I do not like to prolong this discussion and I will only speak for a minute. I have been very much interested in this paper of Dr. Daland. I cannot add anything to it, but I want to stress one or two things which he said.

The first is in relation to colostomy. Whether his figures would be the same in a larger series of cases makes no difference. Apparently the patient who has a colostomy for inoperable cancer of the rectum does not live any longer than the patient who does not. Those of us who have been rather intimately connected with terminal care hospitals, where patients come to die of cancer, are interested in giving the minimum of discomfort rather than in increasing the length of life. I was much interested a few years ago to hear one of the better known English surgeons in the field of cancer of the rectum, say that in their inoperable cases (their operability being between thirty and thirty-five per cent) they did colostomies and the local physician was able to keep the patients very comfortable from then on. I hate to admit that our men here cannot do the same thing.

A colostomy is no joke and I do not think we have any justification for doing a colostomy on a patient with incurable or inoperable cancer of the rectum unless we are going to relieve some specific symptoms which that patient has. A colostomy will

only partly relieve a patient's tenesmus. It will completely relieve the symptoms due to obstruction. Therefore, you must carefully evaluate the symptoms of which the individual patient is complaining before you advise for or against colostomy. I do not believe in a colostomy in anticipation of future obstruction, nor will the patient appreciate that. If on the other hand he has enough obstruction to be pretty uncomfortable, and if you relieve those symptoms, he will like you and like his colostomy.

I would like to say one more thing about a colostomy; too many times the general practitioner gets an improper impression from the patient with incurable cancer. The difference between a colostomy for curable cancer where the growth is removed, and a colostomy without the removal of the growth, is the difference between day and night. They must not be confused.

I was terribly distressed to hear Dr. Allen say he would like to accept a high mortality. I know that Dr. Allen might feel that way, but I know he has lots of friends, and I know he has lots of patients who would not feel that way.

I try very hard to follow all of my patients indefinitely. I have been particularly interested in their reactions to a colostomy. I have felt that it was not quite fair for those of us who are well and normal to pass judgment upon a colostomy but rather to leave this to those who are living with this handicap. When I see these patients bringing up their families, contributing toward their community life, and find them and their families happy, it is very hard for me to go all the way with Doctor Allen when he says that he personally would accept a high mortality in preference to a colostomy.

If I interpret Doctor Daland correctly he says that the young patient with cancer of the rectum lives as long as the older patient with the same condition. This is very important because most of us have had a difficult time in doing very much for young patients with cancer of the rectum. I believe that the reason for this is quite clear. As long as you and I think and talk in terms of a cancer age the average practitioner who sees one case of cancer of the rectum in four or five years will never think of the possibility of cancer in the young patient. In this group a diagnosis of cancer of the rectum is usually made only after the patient has lived long enough to prove every other diagnosis wrong. These patients, then, upon whom a diagnosis should be made at the time of the first visit lose most of their opportunity for cure in the delay which results from our talking in terms of cancer age rather than cancer symptoms.

# DISTRIBUTION OF ACUTE HEAT EFFECTS IN VARIOUS PARTS OF THE WORLD*

BY GEORGE CHEEVER SHATTUCK, M.D.,‡ AND MARGARET M. HILFERTY, ED.M.‡

## Foreword

INITIAL studies having been made of the distribution and probable causes of acute heat effects in the United States† (Shattuck and Hilferty: 1932 and 1933), it seemed desirable to pursue the subject further and to see what could be learned of mortality and acute morbidity from heat in the world as a whole. Such information will make it possible to define more clearly than has yet been done the extent and relative importance of the acute effects of heat in various parts of the world and will serve as a basis for further studies of the causes and means of prevention of such effects in the localities where heat cases are sufficiently numerous to constitute a problem of importance.

There are several circumstances which render such an undertaking difficult. First, no reports are available for some of the places for which one would most desire them. Secondly, deaths from effects of heat being relatively few, some countries report them only as part of the total from external causes, as in the Abridged International List of the Causes of Death. Thirdly, as there are occasional years in which the number of deaths is many times greater than the usual number, an adequate picture can be obtained only from records for rather a long series of years.

It is even more difficult to obtain statistics for morbidity from heat effects. Not being infectious, these cases are not reported to civil health authorities. Morbidity statistics are in hand, however, from two sources; namely, (1) reports of the health of armies and navies; and (2) hospital statistics from parts of Africa. Fortunately, there are "insolation" figures in almost all the army reports which we have seen, even those which list only about thirty causes of sickness. The Annual Report of the Health of the Army of Great Britain is an exception, but the Report of the Public Health Commissioner with the Government of India gives figures for the British Army there which are doubly welcome because of the scarcity of other information from India. We have been able to consult a smaller number of navy records. These army and navy records have the advantage of being practically complete as to cases and deaths, and figures are available as to the mean strength of the forces. However, in general, the deaths are too few to permit of analysis.

The reports of hospitals in certain parts of Africa and in the Far East are the only sources of information obtained from some of these localities.

## Section I

### Mortality Rates per 100,000 by Countries

A summary of the mortality statistics collected from various countries is given in table 1.

*From the Departments of Tropical Medicine and of Vital Statistics of the Harvard School of Public Health.

†Sunstroke and allied conditions in the United States. Am. J. Trop. Med. **12**: 223, 1932.
Causes of deaths from heat in Massachusetts. New Eng. J. Med. **209**: 319, 1933.

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TABLE 1

HEAT DEATHS, DEATH RATES PER 100,000, AND SEX RATIOS BY COUNTRIES

| Country | Years | Total Deaths | Rate per 100,000 | M/F Ratio |
|---|---|---|---|---|
| *Europe* | | | | |
| Finland | 1911-1925 | 15 | .03± .01 | 6.5 |
| Sweden | 1911-1930 | 76 | .06± .01 | 3.0 |
| Norway | 1928-1931 | 6 | — | — |
| England and Wales | 1902-1930 | 3,696 | .34± .05 | 2.0 |
| Irish Free State | 1922-1931 | 32 | .11± .02 | 2.6 |
| France: Paris | 1910-1929 | 78 | .13± .05 | 3.3 |
| The Netherlands | 1916-1930 | 443 | .43± .31 | — |
| | 1901-1930 | — | .25 | 3.0 |
| Germany | 1928-1930 | 302 | .16 | 2.8 |
| Prussia | 1925-1929 | 362 | .19± .04 | 2.6 |
| Berlin | 1900-1917 | 92 | .25± .06 | 4.1 |
| Russia: Kief | 1918-1927 | 4 | — | — |
| Hungary | 1903-1912 | 650 | .31± .08 | 1.8 |
| Switzerland | 1901-1930 | 246 | .21± .03 | 3.6 |
| Czechoslovakia | 1919-1922 | 48 | .09± .02 | 1.5 |
| Greece | 1921, 22, 25, 26 | 57 | .23± .05 | 1.7 |
| Italy | 1911-1928 | 1,653 | .25± .04 | 3.1 |
| Spain | 1901-1915, 1917-1918, 1921-1929 | 1,114 | .21± .01 | 2.0 |
| *Africa* | | | | |
| *Union of South Africa (whites only) | 1913-1918, 1922-1928 | 96 | .50± .06 | 3.6 |
| Mauritius (Indian Ocean) | 1921-1931 | 0 | — | — |
| *Asia* | | | | |
| Ceylon | 1909-1932 | 66 | .06± .01 | 8.0 (1921-32) |
| Straits Settlements | 1922-1931 | 4 | — | — |
| Shanghai (Resident Foreign Community) | 1915-1932 | 29 | 5.8± 1.6 | — |
| Bombay | 1920-1929 | 46 | .4± .1 | — |
| Madras | 1918-1927 | 18 | .3± .1 | — |
| *Pacific Islands* | | | | |
| Japan | 1909-1916, 1918, 20, 21, 1924, 25 | 3,490 | .48± .04 | 1.4 |
| Australia | 1908-1931 | 1,951 | 1.49± .18 | 1.9 |
| New Zealand | 1921-1932 | 17 | .10± .03 | 1.1 |
| *North America* | | | | |
| Canada Registration Area | 1921-1931 | 597 | .65± .17 | 2.0 |
| United States America Registration Area | 1900-1932 | 29,282 | *1.78± .39* | 2.5 |
| Mexico City | 1916-1920 | 13 | — | — |
| *Bermuda and West Indies* | | | | |
| Bermuda | 1922-1932 | 1 | — | — |
| Cuba | 1910-1926 (excluding 1915) | 70 | .2 | 7.8 |
| Haiti | 1925-1929 | 0 | — | — |
| St. Lucia | 1923-1932 | 0 | — | — |
| St. Vincent | 1923-1932 | 0 | — | — |
| Barbados | 1925-1928 | 1 | — | — |
| Grenada | 1912-1931 | 3 | — | — |
| Trinidad and Tobago | 1914-1932 | 3 | — | — |
| Jamaica | — | 94 | .3± .04 | — |
| *Central America* | | | | |
| British Honduras | 1900-1904 | 5 | — | — |
| Republic of Panama | 1920-1928, 1930 | 204 | *4.4± .62* | — |
| *South America* | | | | |
| Colombia | 1915-1920, 1923-1924, 1927-1928 | 604 | .96± .48 | 1.0 |
| British Guiana | 1916-1932 | 13 | — | — |
| Brazil | | | | |
| Rio de Janeiro, F. D. | 1913-1916; 1920-26 | 129 | *1.0± .4* | 3.0 |
| Sao Paulo | 1913-1920 | 0 | — | — |
| Paraguay | 1928-1932 | 2 | — | — |
| Argentina | 1911-1915 | 244 | .62 | 5.3 |
| Uruguay | 1905-1920 | 23 | .10± | 3.6 |
| Chile | 1904-1908, 1911-1930 | 90 | .10± .02 | — |
| Peru: Lima | 1913-1920 | 1 | — | — |

*Table 8.

The tabulations are of the years for which we have information from the various countries or parts thereof. The headings indicate (1) the total number of deaths from this cause; (2) the mean death rates from effects of heat; and (3) the ratio of males to females among the decedents. The mean death rates are given with the standard deviation of the mean based on the computed and not the Bernoulli standard deviation.

The number of deaths in the United States of America in thirty-three years, 1900-1932, is greater than the total of all the others here recorded. The average rate for the United States of America for this period is exceeded, however, by those for the Resident Foreign Community of Shanghai and for the Republic of Panama; and it is approached by the rate for Australia. (See italicized figures in table 1.) The Australian rate is based on mortality in the years 1908-1931. The average rate in the Registration Area of the United States is 1.78± .39 per 100,000 which does not differ significantly from the Australian rate of 1.49± .18. The next highest rates are found in Colombia and in Rio de Janeiro (Federal District), but neither of them is very well determined. Argentina, Canada, the Union of South Africa and Japan have rates of from .48 to .65. The European countries from which we have data except The Netherlands which has a rate of .43± .31, show very moderate rates of less than .35 per 100,000. Only a quarter of the States in the United States of America Registration Area had rates as low as this.

A word of explanation is required about the figures for The Netherlands. The numbers of deaths occurring in each of the years 1916-1930 have been published.* The total is 443 of which 303 occurred in 1923. On this account, the mean rate for the period is not at all representative. Excluding 1923, the average rate for these years is .15, which gives a picture of the usual mortality from effects of heat. But this figure is falsely low and, on the other hand, when 1923 is included, the rate of .43± is misleadingly high. The rates being given by quinquennial averages from 1901-1916, one can obtain an average rate for the thirty years 1901-1930, and thus give less weight to the 1923 figure. Even so, the average rate, .25, is at least twice as high as it would have been without the 1923 experience. This is an extreme example of the difficulty in discussing death rates from heat.

### DEATH RATES IN UNUSUAL YEARS

Certain years stand out as periods in which high rates were rather general. Chief among these is 1911, when England and Wales, Hungary, Italy, Paris, Switzerland and the United States all had large numbers of deaths from heat. The rates differed greatly, being .36 in Italy, 1.49 in England, and 5.3 in the United States; but each was well above the normal. In England, the rate was the highest in the twenty-nine years 1902-1930. The 1911 rate in Italy, however, was exceeded in 1921 and in 1928. The year 1921 brought also a high rate for England and for the British soldiers in India (table 2). In 1928, the civilian mortality in Italy was most severe, and so also was the military morbidity in Germany and that of the French soldiers in Algeria. Elsewhere, the year was not unusual. In 1923, when the death rate in Holland was abnormally high, England experienced its second highest rate.

Inasmuch as our previous studies of heat effects in the United States showed that unusual mortality there was closely correlated with unusually high atmospheric temperatures continuing for several days at least, and because there is no reason to suppose that the normal habits of the people of Europe or of India were disturbed in the years above mentioned, it is highly probable that these unusually high death rates were caused by particularly unfavorable atmospheric conditions and that the chief cause was excessive atmospheric temperatures, perhaps, intensified by high humidity or low wind velocity.

### COMMENTS ON SECTION I

The data in table 1 give but a fragmentary picture of the world-distribution and prevalence of deaths from heat. One may, however, draw the following inferences from the data:

(1) The proportion of deaths from heat in Europe and in the British Isles is much smaller than that in the United States.

(2) Canada has a decidedly smaller proportion of deaths from heat than has the United States.

(3) Apparently, there are few deaths from heat in Mexico City, the West Indies or Bermuda.

(4) The figures for Central America, for South America, for Africa and for Asia, permit of no generalizations. Some of the rates are notably low and others outstandingly high. The striking divergencies indicate the need for further data from these regions.

(5) The sex ratios shown in table 1, with few exceptions, indicate that deaths from heat are far more common in men than in women.

## SECTION II

### HEAT EFFECTS IN VARIOUS ARMIES AND NAVIES

*Comparability.* The morbidity rates for illness attributed to heat in various armies are shown in table 2. The figures are comparable only in a general way because neither the basis of reporting nor the years for which data are

*Statistiek van Nederland. Statistiek van de Sterfte naar den leeftijd en de Oorzaken Dood.

available are the same for all the armies. The Netherlands records the number of soldiers treated; France, Germany and Italy the number of admissions to infirmaries and to hospitals; Belgium and Spain admissions to hospitals; and the other countries, except Czechoslovakia, simply admissions. Czechoslovakia reports for 1921 to 1924 admissions to hospitals and other soldiers in India. The three higher average rates are .99 for the United States Army (all stations); 1.49 for the Japanese Army; and 4.6 for soldiers from the British Isles in India.

The Japanese average rate is based upon only five years, and two of these years (1914 and 1915) were during the World War. The rates in these years were about five times as

TABLE 2

HEAT MORBIDITY RATES PER 1000 FOR VARIOUS ARMIES

| Period | Austria | France | French Army in Algeria | Germany | Italy | Belgium | Russia | Japan | Spain | The Netherlands | Czechoslovakia | Soldiers from British Isles in India | Native Soldiers in India | French Army in Morocco | American Soldiers in the United States |
|---|---|---|---|---|---|---|---|---|---|---|---|---|---|---|---|
| 1900 | 0.2 | | | .30 | .4 | | .04 | | | | | | | | |
| 01 | 0.1 | .38 | .30 | .12 | .4 | | .02 | | | | | | | | |
| 02 | 0.1 | *.49* | .29 | .20 | .5 | | .02 | | | | | | | | |
| 03 | 0.3 | .24 | .23 | | .4 | *.65* | .03 | | | | | | | | |
| 04 | 0.1 | *.53* | .38 | .12 | .3 | .42 | .04 | | | | | | | | |
| 05 | 0.4 | .38 | .30 | .20 | .7 | | .03 | | | | | | | | |
| 06 | 0.2 | *.49* | .28 | .20 | .3 | *2.33* | .02 | | | | | | | | |
| 07 | 0.2 | .20 | .13 | .12 | | .25 | .04 | | | | | | | | |
| 08 | | | | | | | .04 | | | | | | | | |
| 09 | 0.1 | .18 | *.55* | .21 | .3 | .05 | .03 | | | | | | | | |
| 1910 | 0.2 | .16 | *.60* | .16 | .2 | .09 | .03 | | | | | 1.1 | .18 | | |
| *11* | 0.1 | .45 | .23 | .35 | .4 | *.60* | .04 | .95 | .07 | | | 3.5 | .25 | | |
| 12 | | .23 | .13 | .14 | .2 | .04 | .05 | .61 | .02 | | | 2.8 | .49 | | |
| 13 | | .19 | .19 | | .1 | | | .50 | .01 | | | 0.7 | .09 | | |
| 14 | | | | | | | | *2.72* | .01 | | | 1.3 | .16 | | |
| 15 | | | | | | | | *2.65* | .04 | | | 4.6 | .40 | | |
| 16 | | | | | | | | | | | | 5.8 | .31 | | |
| 17 | | | | | | | | | | | | 1.5 | .16 | | |
| 18 | | | | | | | | | .01 | | | *10.3* | *2.24* | | |
| 19 | | | | | | | | | .03 | | | 7.4 | .20 | | |
| 1920 | | .07 | .20 | | | | | | | 0.1 | | 6.1 | .79 | .38 | .80 |
| 21 | | | | | .07 | | | | | | | *19.0* | .50 | | 1.14 |
| 22 | | .11 | .16 | .36 | .06 | | | | .02 | | .05 | 3.4 | .58 | .36 | 1.23 |
| 23 | | .20 | .13 | | *.20* | | | | .02 | *1.2* | .77 | 6.3 | .33 | .92 | .73 |
| 24 | | .28 | .19 | | *.22* | .07 | | | .01 | | .14 | 4.5 | .18 | .29 | 1.21 |
| 25 | | .16 | .26 | .36 | .10 | .21 | | | .04 | *1.0* | .12 | 1.6 | .21 | *1.15* | |
| 26 | | .13 | .12 | .60 | .2 | .11 | | | .01 | .8 | .32 | 3.7 | .20 | .35 | |
| 27 | | .13 | .21 | *.79* | .3 | .15 | | | | .3 | .19 | 4.0 | .50 | .31 | .85 |
| 28 | | .30 | *1.13* | .87 | .4 | .47 | | | .09 | .1 | .36 | 4.3 | .36 | .27 | .82 |
| 29 | | .32 | .32 | .71 | .01 | *.54* | | | .04 | .2 | .46 | 1.7 | .20 | | 1.10 |
| 1930 | | .38 | .32 | .73 | | *.59* | | | .01 | .4 | | 2.3 | .30 | | *1.44* |
| 31 | | | | | | .26 | | | | | | 5.2 | .57 | | 1.17 |
| 32 | | | | | | .44 | | | | | | | | | .43 |
| Adm. rates Average | *.18* | .27 | .30 | .36 | .27 | .43 | .033 | *1.49* | *.024* | .37 | .22 | *4.6* | .42 | .42 | .99 |

cases lasting twenty or more days and ensuing death or discharge but, after 1925, all cases lasting three days or more or causing death or discharge. Even without quantitative allowances (which seem impossible) for these differences, the figures give useful information.

*Comparisons.* The average rates for most of the armies listed range from .024 for Spain to .43 for Belgium. (The figure for Belgium includes an unusually high rate in 1906.) It is noteworthy that within these limits are included also the averages for French soldiers in Morocco and in Algeria as well as that for native high as in the other years and, therefore, the average rate given for Japan is probably higher than the normal.

The average rate of .99 for the American Army in the United States is for the years 1920-1924 and 1927-1931. It includes cases not only among White but also in Colored soldiers. For the period 1927-1931 we have figures for each camp or station in the United States and for stations elsewhere as well. Rates based upon these figures vary in the United States from 1.9 to .3. Rates of 1.3 were shown both for the northern and for the southern central States lying

east of the Mississippi River but the highest rate was for the southern States on the Atlantic Coast. The lowest rate was for the Pacific Coast and that for the Rocky Mountain States was nearly as low. The rate for White soldiers in Panama Canal Zone was 1.3 and the rates for the Philippine Islands and for Hawaii were very low. The average rate for stations in the United States for the period (1927-1931) was .94 while that for the tropical stations mentioned above was .57. The rate for white soldiers in the Philippine Islands was .6. Thus, the comparative frequency of heat effects in the United States is again brought out.

As a rule, the annual fluctuations of rates for most of the armies are not great but there are noteworthy exceptions which are italicized in table 2. Extreme fluctuations are shown for soldiers from the British Isles stationed in India in 1918 and in 1921.

*Deaths.* The procedure in reporting deaths from heat in the various armies differs considerably. The number of deaths recorded is insufficient for useful statistical comparison.

### HEAT EFFECTS IN CERTAIN NAVIES IN RELATION TO STATION

*Data.* The average morbidity rates for the British Navy and for the French Navy, when compared with those for specific "stations", show very striking differences (table 3). In the British Navy the rate for the "East Indies Stations" is extremely high and that for "China Stations" is notably high. The rate for the French Navy in the Indian Ocean is also notably high, but that for Indo-China is not high.

TABLE 3

BRITISH NAVY

Average Morbidity Rates, 1921-1930, by Stations

| Stations | Rates per 1,000 | |
|---|---|---|
| Home | 0.14± | 0.05 |
| Atlantic Fleet | 0.18± | .06 |
| Mediterranean Station | .90± | .11 |
| America and West Indies | 1.2 ± | .42 |
| China | *4.38*± | .51 |
| East Indies | *13.5* ± | 2.1 |
| Africa | 1.33± | .56 |
| Irregular List | *5.50*± | .9 |
| Total Force Average | 1.09± | .06 |

FRENCH NAVY

Average Morbidity Rates, by Stations: 1902, 1904, 1906 to 1911

| Stations | Rates per 1,000 |
|---|---|
| Mediterranée | 0.4 |
| Atlantique | 0.2 |
| Extrême-Orient | 1.0 |
| Océan Indien | *4.2* |
| Pacifique | 1.8 |
| Indo-Chine | 0.7 |
| Station Locale du Sénégal | 1.8 |
| Forces Navales, Maroc | 0.7 |
| Total Force Average | 1.8 |

### COMMENTS ON SECTION II

The data provided in this section amplify to some extent the information as to the distribution and prevalence of heat effects provided in Section I.

The following important facts are shown in table 2:

1. The rates for the American Army in the United States are markedly high.
2. The rates for soldiers from the British Isles in India are extraordinarily high whereas those for native soldiers are comparatively low.
3. The rates for French soldiers in Morocco and in Algeria and those for native British soldiers in India are nearly equal.
4. The data for the British and the French Navies in the Far East seem somewhat discordant.

## SECTION III

### DATA FROM VARIOUS PARTS OF AFRICA

*General Observations.* Some hospital statistics of heat cases in various parts of Africa have been summarized in table 4. The figures are for cases treated in hospitals or dispensaries, that is to say, for "in-patients" and "out-patients". The deaths are given for in-patients only. In some instances the cases are recorded for Europeans and for natives separately but in other instances these groups are not separated.

The validity of admission rates depends upon the degree of accuracy of the figures for population as well as on the completeness of the reporting of cases. The figures for the European population and its cases are, doubtless, much nearer the truth than those for the native populations. The European population is more accurately known and Europeans are more likely to apply for treatment. On the other hand, native populations can seldom be determined accurately and, in general, the natives are so widely distributed that comparatively few of them are within reach of a hospital when acutely ill. Rates for native population should, therefore, be based upon the number of those to whom treatment is available in fact.

Official data from the Belgian Congo take this aspect of the problem into account. The Belgian tabulations are headed "Mouvement Général de la Morbidité et de la Mortalité dans les Rayons d'Actions des Médecins. . . ." The report for the Belgian Congo in 1925 records 3,399 deaths from all causes among the Blacks and a death rate of 14.2 per M. The population within the sphere of action of the physicians must, therefore, have been considered to be 240,000. But, inasmuch

as the total population of the Belgian Congo is about 8,000,000 it appears that morbidity and mortality were recorded for only about one-thirtieth of the population.

Rates for Europeans have not been determined when less than ten cases were recorded.

*Regional data* are presented in tables 5, 6, 7 and 8 for the Belgian Congo, Nigeria, the Anglo-Egyptian Sudan and the Union of South Africa respectively. Inasmuch as few Europeans are treated for heat effects in any year, the fluctuations in their numbers may not be significant.

When the relative proportion of Europeans to Natives (see table 4) is taken into account, table 5 emphasizes the much smaller proportion of cases among natives in the Belgian Congo than among Europeans. As might be expected from its geographical position and considerable altitude, there appear to have been fewer cases in Katanga than in the other Provinces of the Belgian Congo.

In Nigeria (table 6), the great preponderance of cases among Europeans is again shown. There was a marked variation from year to year in the number of Europeans treated.

The outstanding fact for the Anglo-Egyptian Sudan (table 7) is that 104 natives in the year 1932 were admitted to hospitals for heat effects whereas, in other years, the numbers admitted were small. Moreover, the number of European cases treated in 1932 was not unusual. It seems probable that the large number of heat cases among natives in 1932 was not due to unusual heat but rather to some unusual employment involving unnatural conditions of life.

TABLE 4

CASES OF HEAT EFFECTS IN VARIOUS PARTS OF AFRICA AS SHOWN BY HOSPITAL STATISTICS

| Region | Period | Admissions Europ. | Admissions Native | Deaths Europ. | Deaths Native | Population in Thousands Europ. | Population in Thousands Native | Annual Admis. Rates per 1,000 Europ. |
|---|---|---|---|---|---|---|---|---|
| Southwest Africa | 1907/8 } 1911/12 } | 6 | — | — | — | | | |
| Nyasaland Protect. | 1926–31 | *32* | 4 | — | — | 1 | 1,600 | 5 |
| German East Africa | 1903/4 } 1911/12 } | 12 | 4 | 1 | 2 | | | |
| Tanganyika Territ. | 1925–29 | 60 | | 2 | | 8 | 5,000 | |
| Zanzibar | 1927–32 | *65* | | 1 | | 235 | | |
| Uganda Protectorate | 1926–31 | 30 | | 1 | | 2 | 3,500 | |
| Anglo-Egypt. Sudan | 1922–32 | *41* | *149* | 10 | 13 | 5,800 | | |
| Belgian Congo | 1925, 27 | 27 | *39* | 0 | 2 | 25 | 8,500 | 0.5 |
| Cameroon | 1903/4 } 1911/12 } | 5 | 0 | 2 | 0 | | | |
| Nigeria | 1926–31 | *169* | *30* | 2 | 2 | 3 | 19,000 | *9* |
| Togo | 1904/5 } 1911/12 } | 7 | 1 | 0 | 0 | | | |
| Gold Coast | 1926/27 } 1931/32 } | 87 | | 2 | | 2.4 | 3,161 | |

TABLE 5

BELGIAN CONGO: HEAT CASES

*Hospital Data*

| Region | 1925 Europeans Treated | 1925 Europeans Died | 1925 Natives Treated | 1925 Natives Died | 1927 Europeans Treated | 1927 Europeans Died | 1927 Natives Treated | 1927 Natives Died | Total Cases |
|---|---|---|---|---|---|---|---|---|---|
| Province | | | | | | | | | |
| Congo-Kasai | 2 | 0 | 15 | 0 | 3 | 0 | 1 | 0 | 21 |
| Equatoriale | 6 | 0 | 3 | 0 | 6 | 0 | 2 | 0 | 17 |
| Orientale | 4 | 0 | 2 | 0 | 1 | 0 | 15 | 2 | 22 |
| Katanga | — | — | — | — | 3 | 0 | 1 | — | 4 (1 year) |
| Other Cases | — | — | — | — | 2 | 0 | — | — | 2 |
| Totals | 12 | 0 | 20 | 0 | 15 | 0 | 19 | 2 | 66 |

COMMENTS ON SECTION III

1. With the exception of the death rates for Whites in the Union of South Africa (tables 1 and 8), the data presented on heat effects in

TABLE 6

NIGERIA: HEAT CASES

*Hospital Data**

| Period | Europeans | | | | | Non-Europeans | | | | | Total Cases |
|---|---|---|---|---|---|---|---|---|---|---|---|
| | In-Pts. | | Out-Pts. | | | In-Pts. | | Out-Pts. | | | |
| | Adm. | Died | Adm. | | Died | Adm. | Died | Adm. | | Died | |
| | | | M. | F. | | | | M. | F. | | |
| 1931 | 2 | — | 13 | 4 | — | 3 | 2 | 3 | — | — | 25 |
| 1930 | 3 | 1 | 28 | 5 | 1 | 4 | 2 | 2 | 1 | — | 43 |
| 1929 | 8 | — | 26 | 2 | — | 1 | — | 7 | 1 | — | 45 |
| 1928 | 6 | — | 28 | 2 | — | 1 | — | 3 | 1 | — | 41 |
| 1927 | 4 | — | 14 | 1 | — | — | — | | 2 | — | 21 |
| 1926 | 7 | — | 16 | | — | 1 | — | | — | — | 24 |
| Totals | 30 | (1) | 139 | | (1) | 10 | (4) | 20 | | (—) | 199 |

*There are about 2,900 European officials and fifty-four others. The African population of Nigeria is estimated at 19,000,000.

TABLE 7

ANGLO-EGYPTIAN SUDAN: HEAT CASES

*Hospital Data*

| Period | Europeans | | | | Natives | | | | Total Cases |
|---|---|---|---|---|---|---|---|---|---|
| | Males | | Females | | Males | | Females | | |
| | Adm. | Died | Adm. | Died | Adm. | Died | Adm. | Died | |
| 1932 | 5 | 1 | — | — | 95 | 2 | 9 | — | 109 |
| 1931 | 1 | — | — | — | 5 | 3 | — | — | 6 |
| 1930 | 1 | — | 1 | — | 3 | 2 | 3 | — | 8 |
| 1929 | 1 | — | — | — | — | — | — | — | 1 |
| 1928 | 1 | — | — | — | 5 | 1 | — | — | 6 |
| 1927 | 9 | 2 | — | — | 6 | 1 | — | — | 15 |
| 1926 | 5 | — | 1 | 1 | 9 | 1 | — | — | 15 |
| 1925 | 4 | 2 | 1 | — | 3 | — | — | — | 8 |
| 1924 | 8 | 2 | 1 | — | 1 | — | — | — | 10 |
| 1923 | — | — | 1 | 1 | 2 | — | — | — | 3 |
| 1922 | 1 | 1 | — | — | 8 | 3 | — | — | 9 |
| Totals | 36 | (8) | 5 | (2) | 137 | (13) | 12 | (—) | 190 |

*Red Sea Province:* A note in the 1921 Report says that no less than ten cases of heat-stroke with eight deaths occurred in the Red Sea Province in July and August during an exceptionally hot spell of weather. Six of these cases occurred among the European passengers or crew of ships in port.

TABLE 8

UNION OF SOUTH AFRICA

DEATHS FROM HEAT AMONG WHITES

| Period | Deaths | Death Rate per 100,000 | Population in Thousands |
|---|---|---|---|
| 1913-1918 | 30 | .4 | 1,418 (1918) |
| 1922-1928 | 66 | .6 | 1,677 (1926) |

*Deaths by Years and Sex*

| | 1913 | 1914 | 1915 | 1916 | 1917 | 1918 | 1922 | 1923 | 1924 | 1925 | 1926 | 1927 | 1928 | Total |
|---|---|---|---|---|---|---|---|---|---|---|---|---|---|---|
| Males | 5 | 3 | 4 | 2 | 4 | 5 | 6 | 10 | 6 | 9 | 4 | 9 | 8 | 75 |
| Females | 2 | – | – | 2 | 2 | 1 | 2 | 2 | 4 | 4 | 1 | – | 1 | 21 |

Census: 1918—males 727M; females 691M
1926— " 857M; " 820M

Death rates: 0.7 per 100,000 males; 0.2 per 100,000 females

Death ratio of males to females = 3.6

Africa are not directly comparable with those from other parts of the world.

2. The death rates in the Union of South Africa, from heat, based upon the white population (table 8) are considerably lower than those for the United States of America (table 1). The ratio of males to females is 3.6. Thus, the preponderance of deaths of males to females there is even greater than in the United States of America, where the ratio over a period of thirty-one years was 2.5.

3. Admission rates for heat effects among Whites, as shown by hospital statistics, is very high in Nigeria, notably high in Nyasaland, and strikingly lower in the Belgian Congo (table 4). No comparable rate can be figured for the Anglo-Egyptian Sudan because the number of Whites living there is not known to us.

4. Deaths from heat are not numerous even among Europeans in the parts of Africa referred to, but it is evident that the Europeans are far more susceptible to heat than are the Native races.

## Section IV

### Health Reports from French Dependencies in Africa and in Asia

Through the good offices of Dr. F. Sorel, Médecin Général Inspecteur des Troupes Coloniales, Ministère des Colonies, one of us (G. C. S.) was permitted to examine a large number of typewritten reports in the files of the Ministry of the Colonies. These reports were from officials of the Civil Government having administrative duties, from officials of the Medical and Sanitary Service or from Government Hospitals and Dispensaries. Included among them was a single Military Report from French Somaliland.

#### Africa: 1933

*Circonscription de Dakar.* The number of inhabitants was stated to be approximately 70,000 of whom 45,000 lived in the city of Dakar and 25,000 in 14 villages.

Racial classification:

| | |
|---|---|
| Europeans | 5,812 |
| Syrians or Moroccans | 1,877 |
| French half-breeds and foreigners | 2,867 |
| Native races | 58,546 |

There were forty-one deaths during the year among the Europeans. These deaths were classified under thirty-two heads but no death from heat effects was mentioned. Among assimilated races there were twenty-one deaths listed under nineteen heads but heat effects were not mentioned. Neither was heat mentioned as a cause of death among the natives.

*Senegal.* The *Hôpital Colonial de Saint Louis* treated during the year 445 cases in Europeans or in persons belonging to assimilated races. Of these, six died. The number of natives treated in the hospital was 1,306 and many more were treated in dispensaries. Heat is not mentioned as having caused any deaths.

*Colony of Mauritania.* A report on medical aid to the native population puts their number at about 240,000. The report says that nowhere so much as in Mauritania has environment created such distinct morphological types which react diversely to morbid conditions. The report discusses infectious and parasitic diseases, and says that attendance at the dispensaries is large and is increasing. Nothing is said of heat effects.

*French Soudan.* The *Hôpital du Point G* (five kilometers from Bamaku) treated 581 Europeans and 110 natives in 1932 and 419 Europeans and ninety-five natives in 1933. A single case of insolation occurred in a European.

*Niger Colony.* A report states that, in general, the climate is excellent for Europeans and that the natives are not much disturbed by it. Mention is made, however, of a tendency to loss of mental equilibrium by Europeans as the seasons change. Among fifty-two patients hospitalized at Naimey, no cases were attributed to heat, and heat is not mentioned as having caused either morbidity or mortality among Europeans or natives.

*Ivory Coast.* During the year forty-six deaths of Europeans and 1,507 deaths of natives were reported by physicians. Heat is not mentioned as a cause of any of the deaths.

*Dahomey.* Heat is not mentioned as a cause of morbidity or mortality among cases seen by the medical services.

*French Guinea.* Heat effects are not mentioned among the important diseases of natives.

*Gabun and other Colonies.* If any ill-effects of heat were observed by members of the Health Service these cases were so few as to have been included under the head of "maladies diverses".

Reports of health conditions from the *Colony of the Middle Congo*, from the region of *Lake Chad* and from the *Oubangi-Chari Territory* do not mention heat effects.

*French Somaliland.* Cases of illness treated at the *Hôpital Colonial de Djibouti* during the year came not only from the Colony but also from ships. No case of illness due to heat is mentioned in the report of this hospital.

A report of the *Service Médical du Personnel Militaire*, however, says that although the hot season is very uncomfortable, the climate is healthful and well borne by Europeans; and that "coup de chaleur" seldom attacks any except imprudent or plethoric individuals and most often alcoholics. During the year only one European died from heat.

#### Asia: 1933

*Annam.* The numbers of cases treated in hospitals or dispensaries during the year were as follows:

| | European | Native |
|---|---|---|
| Males | 470 | 21,122 |
| Females | 250 | 18,832 |
| Children | 122 | 5,635 |

Three cases of "coup de chaleur" were recorded. All three patients were men. Two were Europeans and the third a native. All three recovered.

*Cochin China.* The number of patients treated in hospitals and clinics during the year was as follows:

| | European | Native |
|---|---|---|
| Males | 600 | 29,409 |
| Females | 576 | 41,623 |
| Children | 267 | 7,229 |

"Coup de chaleur" occurred in four male natives but was not observed in any of the Europeans.

*Tongking.* Many thousands of patients were treated in government hospitals or dispensaries. The morbidity was relatively high from April to October and especially in May. More than 70,000 natives were treated (males 27,501; females 34,633; children 9,049).

Seven heat cases were observed as follows: four men; two women; one child (whites or natives or both?).

*Protectorate of Cambodia* hospital cases:

| | European | Native |
|---|---|---|
| Males | 183 | 6,459 |
| Females | 129 | 3,335 |
| Children | 57 | 562 |

Cases of "coup de chaleur": two males, one a European and one a native. Both patients recovered.

*Laos.* No heat cases were reported. Hospital cases were as follows:

| | European | Native |
|---|---|---|
| Males | 50 | 3,742 |
| Females | 15 | 2,253 |
| Children | 4 | 835 |

### COMMENTS ON SECTION IV

It is evident that ill-effects of heat in the French Dependencies in Africa in the year 1933 were so few as not to constitute a serious problem. Only two such cases with one death were mentioned in the reports examined. Both cases were in Europeans. One case occurred in the French Soudan and the fatal case in French Somaliland.

An official in Paris said that heat cases were common twenty years ago in the French Colonies and that the sun is still feared there but that all persons wear the "casque" today and that, by virtue of the precautions dictated by experience, heat cases have become scarce.

Small numbers of heat cases were reported in 1933 from the French Colonies in Asia. Their numbers were insignificant, however, as compared with the large numbers of patients treated, and still more so with regard to population.

### SUMMARY

All available data having been collected, we have attempted to ascertain the distribution of heat effects and their comparative frequency in different parts of the world. The data obtained are very inadequate for the purpose. It is believed, however, that they justify the following conclusions:

1. The death rates attributed to heat among European residents of Shanghai and among the population of the Republic of Panama are outstandingly high.

2. Heat effects are notably common in the United States and in Australia.

3. Soldiers from the British Isles stationed in India frequently suffer from heat effects.

4. The frequency of heat effects appears to differ markedly in various parts of tropical Africa. Cases appear to be decidedly more numerous in some of the British Colonies (Nigeria, Anglo-Egyptian Sudan, Nyasaland) than they are today in French or in Belgian territory.

5. In the Tropics in general, persons of European race are attacked far more often than are persons of the native races, but the latter are by no means immune. (Anglo-Egyptian Sudan.)

6. Males are far more liable to heat effects than are females.

7. Heat effects are comparatively uncommon in the West Indies, in the British Isles, in Europe, in the Union of South Africa and in Japan.

8. Notably low rates are exhibited for Finland, Sweden, Ceylon, New Zealand and Lima (Peru). Deaths from heat are recorded as *nil* in Mauritius in the Indian Ocean.

9. There are outstanding fluctuations in heat effects in several countries in certain years. To explain these, it may be necessary to localize the cases sharply, to determine the climatic conditions from day to day in the places where the heat effects were most numerous, and to learn whether there had been marked coincident changes in occupation or mode of life among the victims.

10. It has been said that heat cases were common in the French Dependencies twenty years ago but that they have been few in recent years. Reports for the year 1933 from French Dependencies in Africa and in the Far East indicate a striking scarcity of heat cases.

11. The irregular world distribution of acute effects of heat which the data indicate may be, to some extent, fallacious. Incomplete reporting, different methods of recording or of classification, or incorrect figures for population

might cause misleading irregularities in the figures.

12. Some real irregularities of distribution can, probably, be explained as due to climatic factors which are peculiar to restricted localities.

13. It is interesting to note that acute effects of heat are common in some parts of the tropics and not in others; and that the same is true of the temperate zones. These facts indicate the need of further study to explain the distribution of heat cases with reference to probable causes, climatic or other. Work on this problem will be undertaken in the near future.

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# ANESTHETIC EMERGENCIES*

BY URBAN H. EVERSOLE, M.D.†

THERE is no rule by which situations arising during the course of anesthesia can be divided into emergencies and nonemergencies. Many situations may arise that do not within themselves appear to constitute emergencies but if the anesthetist is not prepared to cope with them they may easily develop into real emergencies.

## PREVENTION OF EMERGENCIES

Undoubtedly the most satisfactory method of dealing with an emergency is its anticipation with the consequent taking of steps for its prevention. An emergency may often be anticipated from a consideration of the condition of the patient or the type of operation.

### THE PATIENT

1. Obviously patients who are poor risks either from debility, age, or long-standing infections such as tuberculosis, as well as those suffering from severe cardiac lesions, are more likely to give difficulty during the course of an anesthesia.

2. Serious obstruction is to be anticipated in patients who have lesions of the neck or throat causing tracheal compression or deviation. When this condition is suspected x-rays should be taken previous to the anesthesia to determine the degree of tracheal abnormality. Adenomatous goiters of the intrathoracic type constitute a large portion of the above group.

3. Patients with intrathoracic exudative lesions such as lung abscess, active tuberculosis, and bronchiectasis often have a great deal of difficulty due to the exudate, which may be aspirated into the good lung and thus result in spreading of the infection or the actual drowning of the patient. If this type of patient is operated upon, intratracheal anesthesia should be used and adequate facilities for aspiration of the air passages should be available at all times.

4. Chronic alcoholics constitute a group which may give the anesthetist a great deal of difficulty because of their great resistance to anesthetic agents, particularly ether. They require heavy preoperative medication and are poor subjects for inhalation anesthesia. When the condition of the patient and the type of operation warrant its use, spinal anesthesia is preferable in these cases.

### THE OPERATION

1. Operations on the neck where there are manipulations around the trachea often cause serious obstruction from a reflex spasm of the vocal cords. This is caused by stimulation of the surface of the trachea or of the laryngeal nerves. Treatment of this condition is discussed later under Mechanical Obstructions of Respiration.

2. Severe drop in blood pressure is to be anticipated when, during the course of an operation, a marked shifting of the position of the patient is necessary, as in the change to the Sims' (or even worse, the prone position) from the dorsal recumbent position in an abdominoperineal resection of the rectum. The administration of .2 to .4 cc. of a 1 to 1000 solution of adrenalin intramuscularly five minutes before the change in position often is of value in preventing such a radical drop.

3. In a thoracic operation respiration is greatly hampered due to the position of the patient on the table. The good lung is on the dependent side and the chest wall often splinted by braces, thus greatly limiting chest expansion and making it very difficult to maintain surgical anesthesia and adequate oxygenation with such weak anesthetic agents as nitrous oxide or ethylene. This difficulty is overcome by the use of cyclopropane[1].

Respiration becomes a greater problem in these operations if the pleural cavity is open. However, when one pleural cavity only is open spontaneous respiration usually continues uninterrupted, particularly if the opening is large. A small opening may cause more air to be drawn in than is expelled with a consequent compression and mediastinal shift. Of course the accidental opening of both pleural cavities necessitates immediate artificial respiration, which can very easily be carried out by means of rhythmic manual pressure on a rubber breathing bag if a closed type of inhalation anesthesia

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is being used. Pressure equivalent to seven to eight millimeters of mercury is usually sufficient to prevent collapse of the lung while 22 mm. should not be exceeded. With a mobile mediastinum sudden opening of a pleural cavity may cause a patient to go into collapse due to flapping of the mediastinum. We feel that all intrapleural operations should have intratracheal anesthesia.

MANAGEMENT OF EMERGENCIES

General Classification of Anesthetic Emergencies

1. Respiratory.
2. Circulatory.
3. Miscellaneous.

I. Respiratory Difficulties

(a) Mechanical obstruction of respiration.
(b) Disturbances of the respiratory center.
(c) Miscellaneous respiratory difficulties.

(A) MECHANICAL OBSTRUCTIONS

Any type of respiratory obstruction, however slight, may become a serious complication during the course of anesthesia and an immediate attempt should be made to rectify it.

(1) One of the most common causes of respiratory obstruction is laryngeal spasm, a condition in which the vocal cords are in adduction, either partially or completely obliterating the air passage. This can be caused by irritation due to the anesthetic agent, reflexly by stimulation of the trachea or laryngeal nerves, or during an abdominal operation under light anesthesia by stimulation of the splanchnics or traction on the mesentery[2].

In most instances the administration under slight pressure (about 10 mm. of mercury) of high concentration of oxygen with a small amount of carbon dioxide is sufficient to relieve the spasm. If the spasm is due to tracheal or laryngeal nerve stimulation it may be necessary to stop the operation for a short time so that the obstruction can be cleared. Occasionally it may be necessary to force an intratracheal catheter between the cords to relieve the obstruction and in rare cases a tracheotomy may be necessary.

(2) Obstruction to respiration may be due to outside pressure on the trachea from a neck tumor such as an intrathoracic goitre causing deviation and compression of the trachea. Any case in which there is actual compression of the trachea (and this can be determined previously by x-ray) should have an intratracheal catheter in place before the operation begins. We have found the flexible metal catheter designed by Flagg and later modified by Woodbridge[3], of this Clinic, to be very satisfactory because of its extreme flexibility and resistance to compression.

(3) Common types of obstruction, usually of minor consequence (though they may become serious), are those due to the dropping back of the tongue under anesthesia, tight compression of the lips, or flutter of the relaxed soft palate or alae nasi. These can usually be relieved by the use of either nasal or oral breathing tubes or by forcibly extending the chin.

(B) DISTURBANCE OF THE RESPIRATORY CENTER

(1) Pathological from increased intracranial pressure

(2) Drug depression divided as follows:

(a) Too heavy preoperative medication with a depressant drug such as the opiates and the barbiturates causes respiratory depression. The depression may be sufficiently great to result in cessation of respiration before anesthesia of sufficient depth is reached. This is particularly true when a respiratory depressant anesthetic such as cyclopropane is being used. This difficulty can usually be overcome by the administration of small amounts (200 to 300 cc. per minute) of $CO_2$ for a short time. For cases in which cyclopropane is to be used we reduce the preoperative medication, our average dose being morphine sulphate grains 1/8 and scopolamine hydrobromide grains 1/150 given subcutaneously one hour before operation, whereas with ethylene or nitrous oxide we usually give 1/6 grain of morphine and 1/150 grain of scopolamine and in addition three grains of nembutal by mouth one and one half hours before operation. These doses vary according to age, weight, and general condition of the patient.

(b) Paralysis of the respiratory center with cessation of respiration of course occurs when an inhalation anesthesia is carried to too great a depth (4th stage of anesthesia). If this inadvertently occurs, administration of the anesthetic agent should immediately be stopped and oxygen with $CO_2$ administered by means of artificial respiration.

(c) Avertin anesthesia is frequently accompanied by a marked degree of respiratory depression which may persist for several hours following the operation. The period of postanesthetic depression may be materially shortened by the administration of 1 to 5 cc. of coramine intravenously. We have found this drug to be of greater value than the more commonly used respiratory stimulant, caffeine sodium benzoate.

(d) A spinal anesthetic drug may reach too great a height causing respiratory depression or even complete respiratory paralysis. The first sign of this is a paralysis of the intercostal muscles, beginning with the lower and working upward, characterized by lack of thoracic expansion and in severe cases even by a sucking in of the chest wall during inspiration, leaving only the abdominal type of breathing. Finally the diaphragm itself may be paralyzed rendering spontaneous respiration impossible. Should this occur, oxygen must be administered by means of artificial respiration with a rubber breathing bag. We have had this occur in two instances. Artificial respiration was immediately instituted and maintained until the patient was able to breathe spontaneously. Both patients had an entirely uneventful convalescence.

(c) MISCELLANEOUS RESPIRATORY DISTURBANCES

1. Opening of Pleural Cavity—already discussed under The Operation.

2. *Hole in the Trachea.* During the course of an operation in the neck such as thyroidectomy there may be an accidental puncture of the trachea. If this accident occurs the surgeon should immediately inform the anesthetist who should at once institute positive pressure to prevent the aspiration of blood into the air passages while the opening in the trachea is being closed.

II. CIRCULATORY EMERGENCIES

a. Cardiac failure.
b. Circulatory failure.

Differentiation of cardiac failure and severe circulatory failure is often difficult.

(A) CARDIAC FAILURE

Fortunately primary cardiac failure under anesthesia is a rare occurrence. When it does occur, it is in all probability due to the direct toxic action of the anesthetic drug itself. This is particularly true of the more toxic agents such as ethyl chloride and chloroform. Cases of primary cardiac failure have been reported in healthy young adults under light chloroform anesthesia. The treatment of this condition is very unsatisfactory. The ordinary measures for the treatment of collapse such as artificial respiration with oxygen should of course be instituted and in addition the circulatory stimulant drugs such as intravenous adrenalin and ephedrin should be used. Adrenalin may also be given directly into the heart. A moderate degree of Trendelenburg position is also of value in combating the secondary cerebral anemia.

(B) CIRCULATORY FAILURE

Circulatory failure occurring during the course of anesthesia may be of two types:

1. Failure due to blood loss or surgical trauma (shock).

2. Circulatory depression due to spinal anesthesia.

SHOCK

Circulatory failure due to loss of blood or surgical trauma is characterized by a rapid, perhaps thready pulse, and a fall in blood pressure. These are frequently accompanied by other signs of collapse such as profuse sweating and respiratory depression. If the patient is not under general anesthesia, an expression of anxiety or a lapse into unconsciousness is not uncommon. Unlike the depression due to spinal anesthesia which may occur quite early, shock is usually not manifested until later in the operation.

The most important therapeutic measure in combating surgical shock is the restoration of fluids, which can temporarily be accomplished by the intravenous administration of a solution of 10 per cent glucose. (Five hundred to one thousand cc. is usually sufficient.) Blood transfusion is of great value in this condition and is employed in any case in this Clinic where any definite degree of shock is present. A moderate degree of Trendelenburg position (10°) is of value only in combating the secondary cerebral anemia.

An attempt should be made to conserve the body heat by the use of blankets and a warm room. The stimulants such as coramine and caffein sodium benzoate and sedatives such as morphine are to be used only as indicated.

CIRCULATORY DEPRESSION (SPINAL)

Though depression due to spinal anesthesia may appear at any time during the course of anesthesia, frequently it is manifested quite early. It is characterized by a falling blood pressure which may or may not be accompanied by a rapid pulse. The patient frequently appears dusky, indicative of an inadequate circulation. Signs of respiratory embarrassment or depression may be present and the patient may become quite apathetic or even unconscious.

Severs and Waters[4] believe that one of the prime factors in circulatory depression in spinal anesthesia is a cellular oxygen want. By experimentation they were able to demonstrate with high spinal anesthesia with some intercostal paralysis, that the fall in blood pressure tended to be synchronous with and proportional to the degree of intercostal paralysis and that the circulation was improved with the administration of oxygen. In dogs they were able by means of artificial respiration to maintain a normal blood pressure after complete transection of the cord at the level of the seventh cervical seg-

ment. CoTui believes that the circulatory depression of spinal anesthesia characterized by low blood pressure "resembles low blood pressure in other states, having the main features of shock, among them a condition of stagnant anoxia". He believes this depression is due to the loss of central action as a result of direct action of the anesthetic drug itself on the circulatory center in the medulla[5].

Regardless of the method, the primary aim of any treatment of circulatory depression should be the restoration of adequate cellular oxygen. A moderate degree of Trendelenburg position (10° usually sufficient) is of value in increasing the circulation to the upper part of the body, particularly the brain. Extreme Trendelenburg position should not be used as it tends to throw the weight of the viscera against the diaphragm, thereby embarrassing respiration.

Oxygen should be administered by means of a gas anesthesia apparatus. In cases which present respiratory depression, small amounts of $CO_2$ (5 to 10 per cent) should be added and the patient may be assisted in breathing by a small amount of pressure on the breathing bag during inspiration.

There is a considerable diversity of opinion as to the value of the constrictor drugs. Severs and Waters[4] as well as Sise[6] feel very definitely that the constrictor drugs are of great value in the treatment of the circulatory depression of spinal anesthesia, if given only in doses sufficient to restore normal blood pressure. Severs and Waters tend to favor ephedrin. Because of its slow action ephedrin should be given intravenously. Sise on the other hand favors adrenalin given intramuscularly except in very severe cases when it is given intravenously. The anesthetist should always bear in mind that the treatment of a collapsing circulation is much more satisfactory than that of a collapsed one. The administration of ephedrin in 50 mg. doses 5 to 10 minutes before spinal anesthesia is given is an aid in the prevention of a severe blood pressure fall.

Regardless of the type of anesthesia, whether regional, spinal or general an experienced anesthetist should be in constant attendance on the patient.

In general the following plan of treatment is used in this Clinic in cases of circulatory depression.

(1) Careful watching of the patient for early signs such as rapid or labored breathing, sweating, slight degree of cyanosis, rapid pulse or an unusually slow pulse, or falling blood pressure.

(2) Administration of oxygen by means of gas machine, using artificial respiration if necessary.

(3) Adrenalin in doses .2 to .4 cc. of 1 to 1000 solution intramuscularly with massage at the site of injection; this dose is to be repeated in fifteen minutes if response is not adequate. (Attempting to maintain the blood pressure at least 2/3 preanesthetic level.)

(4) Ten degrees Trendelenburg position except in very obese patients.

(5) Administration intravenously of fluids (500 to 1000 cc. of 10 per cent glucose) if there has been considerable blood loss and in addition an immediate blood transfusion if the patient's condition is considered grave enough to warrant it. One should always bear in mind that there is danger of pulmonary edema in the administration of large quantities of fluid to patients with damaged circulation.

### III. Miscellaneous Emergencies

(A) *Inadequacy of Spinal Anesthesia.* The inadequacy of spinal anesthesia, usually due to insufficient height or longer operating time than anticipated, necessitates the addition of a general anesthetic. This is fraught with certain dangers, particularly if an ether anesthesia follows a spinal. Before the advent of cyclopropane, it was often necessary in these cases to employ ether to provide adequate relaxation. The anoxemia which so frequently accompanies nitrous oxide anesthesia makes this gas less satisfactory as a supplement for an inadequate spinal. With the advent of cyclopropane we now have a drug definitely less toxic than ether, capable of administration in relatively low concentration (under 25 per cent), affording relaxation approaching that of ether and without any danger of anoxemia.

(B) *Spinal Too High.* The treatment for circulatory and respiratory disturbances due to the spinal anesthetic drug reaching a height sufficent to cause respiratory depression has already been discussed under Respiratory Emergencies.

(C) *Nausea—Retching—Vomiting.* Retching and vomiting are frequent occurrences under spinal anesthesia. They are quite distressing to the patient and they also make the surgeon's work in the abdomen almost impossible during the spasm. Surgical manipulations probably account for most of the retching and vomiting though CoTui[5] thinks this condition is due to direct action of the anesthetic agent on the vomiting center, hence should be taken as a warning sign by the anesthetist.

The treatment of this condition is frequently most unsatisfactory. Since the psychic element in many instances plays a large part in the initiation of spasms of retching and vomiting, reassurance of the patient by the anesthetist that everything is all right, may be helpful. Instructions to breathe deeply through the mouth a few times and the application of cold cloths to the forehead and neck will often prevent or

shorten the spasm. Inhalations of oxygen are sometimes helpful though if the patient is wide-awake and inclined to be nervous the placing of a mask over his face frequently disturbs him more. If the condition persists, a light gas anesthesia should be given. Cyclopropane with its high oxygen content and ease and rapidity of induction has proved very satisfactory for these cases.

Our clinical impression is that there is no marked difference in the amount of nausea and vomiting during the operation between the patients who have had heavy and those who have had light preoperative narcosis.

(D) *Convulsions.* Convulsions are an infrequent occurrence under any type of anesthesia. The most common cause is the inadvertent intravenous administration of a local anesthetic drug such as novocain although an occasional case may occur in the course of ether anesthesia.

The two main points in the prevention of convulsions from inadvertent intravenous novocain administration are as follows:

1. Repeated aspirations during the course of injection of a local anesthetic or the frequent moving of the needle during the course of an extensive infiltration so that more than 2 cc.'s are never injected at the same site.

2. The administration preoperatively of a barbiturate such as nembutal (pentobarbital sodium) or sodium amytal.

Should a convulsion occur the immediate intravenous administration of sodium amytal (3 to 12 gr.) or of nembutal (1½ to 3 gr.) will usually relieve the condition.

(E) *Broken Needles.* Fortunately the breaking of needles in the spine is a rare occurrence. Its incidence is greatly decreased by the use of needles made of a noncorrosive metal. At the Lahey Clinic a 21 gauge gold needle is used for all spinal anesthesias. We have never had the shaft of a gold needle break.

Unless the fracture in the needle is sufficiently superficial to permit the grasping of the broken segment with forceps, it becomes of necessity a surgical problem and an immediate surgical attempt to remove the broken portion should be made. Several cases where the spinal needle has been broken off in the canal have been sent to the Clinic for removal and a method for their removal has been described by Dr. Lahey of this Clinic[7].

SUMMARY

The more common emergencies with which the anesthetist has to cope and the methods for treatment which we now feel to be most satisfactory, have been presented.

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# THE RELIEF OF PAIN BY THE SUBARACHNOID INJECTION OF ALCOHOL*

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THE inevitable demand for the relief of pain caused by cancer has resulted in the development of a number of palliative neurosurgical procedures. Chief among these are chordotomy, posterior root section, and paravertebral alcohol injection. None of these procedures have been widely employed. Chordotomy and posterior root section not only require the services of a highly trained neurosurgeon, but they are totally inapplicable to the patient near death from carcinomatosis. Paravertebral alcohol injection is time-consuming, technically difficult, sometimes causes a painful neuritis, and often provides only transitory relief. Consequently, the majority of surgeons have been forced to rely upon the liberal use of morphine for the relief of intractable pain.

Several years ago Dogliotti[1] described a method of dealing with intractable pain which is deserving of careful consideration because it is so simple technically that its execution falls within the domain of the general surgeon. This method is the subarachnoid injection of ethyl alcohol which, being lighter than cerebrospinal fluid, should float upon it. Thus, by keeping the affected side of the patient uppermost, and by flexing the spine laterally (fig. 1), it is possible theoretically to bring the alcohol into contact with the dorsal roots of the affected side at any desired level. However, the exact site of the action of the injected alcohol is not known. Dogliotti supposed that the alcohol destroyed a number of nerve fibers in the dorsal

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root without completely blocking it. Stern[2] also believes that the alcohol acts directly on the dorsal root, and that by adjusting the position of the patient so that the spine is rotated forward to an angle of forty-five degrees (fig. 2), it can be brought into contact with the

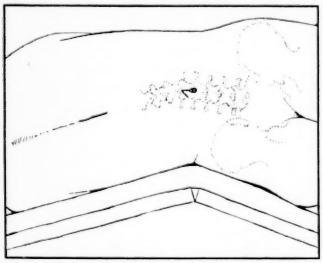

FIG. 1. Patient in position for alcohol injection of right twelfth thoracic interspace. Slight forward rotation of the spine, usually employed, is not shown.

dorsal roots without affecting the anterior roots. It seems unlikely that the position of a patient can be so accurately controlled that one or two cubic centimeters of alcohol floating on the spinal fluid can be made to affect only the posterior nerve roots. Motor nerves are much more resistant to anesthetic agents than sensory nerves[3, 4], and we believe that this fact accounts for the absence of motor paralysis following subarachnoid alcohol injections. Moreover, the action of the alcohol is not confined to the dor-

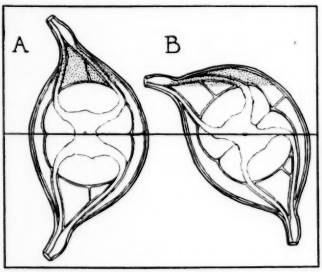


FIG. 2. a. Locus of action of alcohol with patient in position as shown in fig. 1.
b. Theoretical locus of action when spine is rotated slightly forward showing the alcohol in contact with the posterior root only (after Stern). N. B. The authors are not convinced that this theoretically ideal position can be obtained with certainty (see text).

sal roots, as postmortem studies in one of our cases have shown definite degenerative changes in the nerves of the cauda equina.

The mechanism of the action of the injected alcohol is also not known. There is disagreement among investigators regarding the action of alcohol on the living nerve[5], and even less is known about the action of alcohol injected into the subarachnoid space. Postmortem studies in two of our cases were not very enlightening. In one case, examination made eight days after an injection, which had given very effective relief of pain, and also had caused complete urinary retention, showed no significant changes. In the other case, in which several repeated injections were made, there were degenerative changes in the cauda equina manifested by vacuolization of nerve cells and mononuclear infiltrations. Similar, but less marked changes were also seen in the posterior nerve roots and in the cord.

It would seem that the mode of action of the injected alcohol is as obscure as that of the commonly used spinal anesthetics. Moreover, in our experience it is not always possible to limit accurately the localization and action of the injected alcohol to particular nerve segments. Theoretically, the alcohol should be injected at the level of emergence of the nerve root from the cord, which is slightly higher than the corresponding vertebral interspace (fig. 3), and, by slightly flexing the spine laterally, limit the action of the alcohol to that segment and the adjoining ones. As can be seen from a study of table 1, this, although reasonable in theory, does not work out in practice. In some cases an injection in the third lumbar space has given an excellent anesthesia involving nerves as high as the first lumbar and twelfth dorsal nerves, and vice versa. We believe, however, that it is advisable to make the injection at the highest segment involved. Then if the anesthesia produced affords only partial relief a careful sensory examination will show that only a portion of the painful area has been blocked, and that reinjection at a lower level is necessary.

It can be seen from figure 5 that if the anesthetic area coincides with the painful area, satisfactory relief can be expected in practically every case.

## TECHNIC

A technic similar to that recommended by Dogliotti[1] was used. However, following the suggestion of Dr. Tracy Putnam, we used a mixture of sixty per cent absolute ethyl alcohol and forty per cent absolute methyl alcohol in all but two cases. In these two cases absolute ethyl alcohol was used. We have employed dosages considerably larger than those recommended by Dogliotti or Stern. Lumbar puncture was performed in the interspace corresponding to the level of emergence from the cord of the nerves supplying the painful area. Segmental charts are useful in selecting the site of injection. (Figs. 3 and 4.) It should be noted, however, that, as can be seen in case 1, an excellent result may be obtained even when

the alcohol is injected slightly below the level of emergence of the involved roots. In view of the pathological findings in case 13, it seems

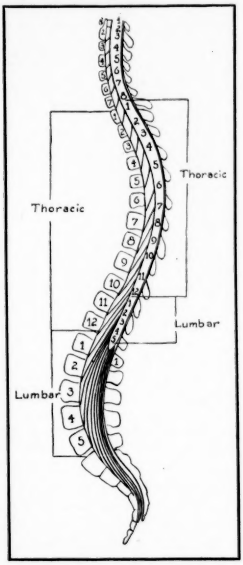


FIG. 3. Note the difference between the level of emergence of the nerve roots from the cord, and the level of the corresponding vertebral interspace (after Ranson).

likely that paralysis of the sensory fibers in the cauda equina may account for successful results when the injection is made below the supposedly optimum site. A number nineteen gauge needle was used and in our later experience an amount of spinal fluid equivalent to the amount of alcohol to be injected was withdrawn. The head of the patient was kept at a lower level than the site of injection and the spine was flexed to as acute an angle as possible at this point (see fig. 1). Eight-tenths to two cubic centimeters of the alcohol mixture was slowly injected, taking at least one full minute for each cubic centimeter. In cases in which sacral nerve segments have been involved we have kept the entire lower lumbar and sacral segments elevated above the site of injection. This procedure is carried out on an operating table, which greatly facilitates adjusting the position of the patient. Occasionally it may be more convenient to make the injection with the patient in bed. Following the injection the patient is kept as nearly as possible in the same position for four hours. Afterwards he should be kept flat in bed for twelve hours and confined to bed for several days. Attempts to get patients up sooner have caused distressing headaches. We have found the preoperative administration of phenobarbital, 200 milligrams, and morphine, 10 to 15 milligrams, advantageous in keeping patients comfortable during the period following the injection at which time there usually occurs an uncomfortable burning sensation which is always transient.

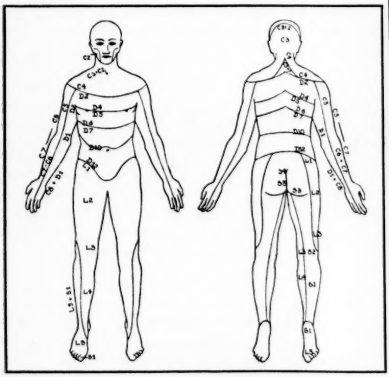

FIG. 4. The segmental innervation of the body.

The dosage must be determined by the conditions existing in each individual case. The larger amounts of one and one-half to two cubic centimeters allow the alcohol to affect a wider area at one injection. In our experience no serious complications have followed the use of the mixture of ethyl and methyl alcohol in doses up to two cubic centimeters. On the other hand, in one case in which two cubic centimeters of absolute ethyl alcohol was used, acute retention of urine developed and persisted until death. It is far better to err on the side of conservatism as reinjection can be performed easily, and serious complications may follow large doses. The maximum dose, therefore, should not exceed two cubic centimeters of the mixture of ethyl and methyl, or one cubic centimeter of ethyl alcohol.

## RESULTS

Yeomans[6], Saltzstein[7], and Stern[2] have reported satisfactory results from the subarachnoid injection of alcohol. During the last two years it has been employed in a selected group of cases on the urological and surgical services of the Peter Bent Brigham Hospital. Although the number of cases is small it seems worth while to report the results, as the collective experience of different investigators leads to the more rapid acceptance or rejection of such a technically simple but potentially dangerous procedure.

Table 1 is a summary of our results to date.

In six cases the result was very satisfactory; in five cases there was either definite improvement without complete relief, or marked improvement, but with rapid recurrence of the pain. Two cases were accounted as failures. The satisfactory results were obtained in cases of carcinoma of the cervix, bladder, testicle and prostate. Improvement without complete relief occurred in the same type of cases.* In general, the more extensive the lesion the less satisfactory was the result. This can be seen from table 1 and figure 5, which show that the more nearly the anesthetized area coincided with the painful area as mapped out on the skin, the more successful was the result. Bilateral cases were particularly disappointing. In the cases (1, 4, 12) in which the pain was referred to a peripheral somatic nerve (sciatic), dramatic relief was obtained. When the pain was more deep-seated and less sharply localized, often being heavy and boring in character, the results were much less satisfactory. It is our impression that so-called visceral pain is less amenable to this form of therapy than is somatic pain, probably due to the fact that, as Davis[8] has emphasized, the abolition of visceral pain requires the section or blocking of a large number of posterior roots.

However, even in the cases in which we have not obtained an entirely satisfactory result the measure of relief obtained has been so appreciated by patients and relatives that we felt the injection was worth while. Of the two cases that are accounted as failures, one was a patient with cancer of the bladder who had a large sacral cyst which may have communicated with the subarachnoid space. At any rate, three injections by two different surgeons produced no anesthesia. The other case was that of a neurotic woman who complained of pain in the distribution of the ilioinguinal nerve following nephrectomy. Although definite anesthesia was obtained over the course of the nerve the patient continued to complain of pain. In retrospect we feel that the selection of this case was ill advised not only because of the neurotic nature of the patient, but because of the benign character of the lesion.

In successful cases the relief of pain is extraordinary. In our cases the pain disappeared dramatically within a few minutes or an hour after the injection, although in one case it was delayed for almost a week. Dogliotti and Stern have noticed this occasional delayed effect also, but in our case (case 11) it may have been due to roentgen-ray therapy rather than to the delayed effect of the alcohol. Our observations confirm those of Dogliotti in regard to the character of the anesthesia. It is as if nerve filaments rather than an entire nerve were blocked. Thus, in our most successful case in which dramatic relief of pain was obtained for nine months, the patient was able to appreciate a stout pinprick in certain portions of the anesthetic area, light touch was not entirely abolished, and temperature sensation was hardly affected. Yet the threshold of pain was raised sufficiently to provide complete relief. In other cases, particularly those in which large dosages were used, pain, temperature, and tactile sensation have been completely abolished and reflex activity greatly diminished.

*We have had no experience with lesions involving areas above the level of the twelfth dorsal nerve, but injection may be made as high as the first thoracic segment[2].

### COMPLICATIONS

Although the subarachnoid injection of alcohol is still in point of fact a clinical experiment without complete laboratory investigation to guide it, serious complications have not been common. Theoretically a respiratory paralysis may occur from a rapid diffusion of the drug upwards in the cord. It has not been reported and we have had no difficulty in this respect. If care is exercised in keeping the patient's head lower than the site of injection this complication should be impossible. Motor paralysis and loss of sphincter control of the bladder and rectum may occur. Permanent motor paralysis has not occurred although transitory weakness is common. It was seen in seven of our thirteen cases. Sphincter paralysis occurred in one case in which two cubic centimeters of absolute ethyl alcohol were used. It was characterized by acute retention of urine persisting until death ten days later. Stern[2] feels that paralysis of the sphincters can be avoided if doses of not more than eight-tenths of a cubic centimeter are used for injections low in the cord. This applies to absolute ethyl alcohol, but we feel it is safe to use the mixture of ethyl and methyl alcohol in doses up to one and one-half cubic centimeters at low levels. Meningismus occurred in two cases persisting for several days after the injection. Examination of the spinal fluid five days after the injection in one of these cases showed a slight increase in total protein and lymphocyte count.

### FAILURES AND REINJECTIONS

Injections have been repeated in four of our cases excluding the two in which bilateral injection was done. As has already been emphasized, in practically all cases in which relief of pain was not obtained the anesthetic area did not coincide with the painful area (fig. 5). We attribute this partly to our inability to control accurately the localization of the injected alcohol, but also, to an inevitable defect in the method, for, in order to block an extensive group of nerves it is necessary to use amounts of alcohol in excess of the margin of safety. For this reason we prefer the mixture of ethyl and

TABLE 1

| | Lesion | Distribution of Pain | Site of Injection and Amount of Alcohol | Anesthetic Area | Site of Reinjection and Amount of Alcohol | Anesthetic Area After Injection | Final Result | Complications | Comment |
|---|---|---|---|---|---|---|---|---|---|
| (1) | Carcinoma cervix. | Right leg—area supplied by nerves $L_3$, $L_4$, $L_5$, $L_6$. | 3rd lumbar interspace. 1 cc. alcohol mixture. | $L_5$, $L_6$. | 4th interspace. 1.5 cc. alcohol mixture. | $L_3$, $L_4$, $L_5$, $L_6$. | (1) Partial relief. (2) Complete relief for nine months; death by lobar pneumonia. | None. | Excellent. |
| (2) | Carcinoma cervix. | Right sacro-iliac region. Vesico-vaginal fistula. Area supplied by $L_4$, $L_5$, $S_1$. | 3rd lumbar interspace. 1.5 cc. alcohol mixture. | $L_5$, $L_6$. | 4th interspace. 2 cc. alcohol mixture. | $L_4$, $L_5$, $L_6$. | (1) Partial relief. (2) Partial relief. | None. | Fair improvement without complete relief. |
| (3) | Carcinoma cervix. | Abdomen, right buttock and leg. Area supplied by $D_{10}$, $D_{12}$, $L_1$, $L_2$, $L_3$, $L_4$, $L_5$. | 3rd lumbar interspace. 1 cc. alcohol mixture. | $D_{12}$, $L_1$, $L_2$, $L_3$, $L_4$, $L_5$, $S_1$. | None. | | Complete relief of pain in leg for four months. Later developed severe pain in back after discharge from the hospital. | Motor weakness. Meningismus four days. | Very good, relatives grateful for measure of relief obtained. |
| (4) | Carcinoma cervix. | Vesicovaginal fistula. Lower abdominal pain. Area supplied by $D_{10}$, $D_{12}$ bilaterally. | 1st lumbar interspace, left. 2 cc. alcohol mixture. | $L_1$, $L_2$, $L_3$, $L_4$, $L_5$. | 1st lumbar interspace right. 2 cc. alcohol mixture. | Both legs. $L_1$, $L_2$, $L_3$, $L_4$, $L_5$. | Very little improvement. | None. | Poor result may be due to low injection, might have been repeated with profit. |
| (5) | Carcinoma cervix. | Right sacro-iliac joint. Area supplied by $L_4$, $L_5$, $S_1$. | 12th thoracic space. 1.5 cc. alcohol mixture. | $L_3$, $L_4$, $L_5$, $L_6$. | None. | | Immediate complete relief, four weeks. Pain recurred four weeks later in sacral area which was not blocked. | Motor weakness. | Recurrent pain in back in sacral area which was not blocked. |
| (6) | Carcinoma prostate. | Lower lumbar and sacral regions. Area supplied by $L_4$, $L_5$, $S_1$, $S_2$. | 2nd lumbar space. 2 cc. alcohol mixture. | $S_1$ only. | None. | | Definite improvement, but recurrence of pain later. | None. | Fair. Pain persisted in sacral area which was not blocked. |
| (7) | Carcinoma prostate | Left leg—area supplied by $L_4$, $L_5$, $L_6$, $L_6$. | 4th lumbar interspace. 0.8 cc. alcohol mixture. | Not recorded. | None. | | Excellent relief. Death one week later. | None. | Excellent until death. |
| (8) | Carcinoma prostate. | Right lower quadrant just above inguinal ligament. Area supplied by $D_{10}$, $D_{12}$, $L_1$. | 1st lumbar interspace. 2 cc. alcohol mixture. | $D_{12}$, $L_1$, $L_2$, $L_3$, $L_4$, $L_5$, $S_1$. | None. | | Excellent relief—no recurrence of pain when last seen seven months later. | Motor weakness. | Excellent. Patient also had roentgen ray therapy. |
| (9) | Neuritis. | Ilio-inguinal nerve, postnephrectomy. | 12th lumbar interspace. 1 cc. alcohol mixture. | $D_{12}$, $D_{12}$, $L_2$. | None. | | Good immediate relief—no apparent relief after twenty-four hours. | None. | Failure. |
| (10) | Carcinoma bladder. | Lower abdomen and both legs. | 1st and 2nd lumbar interspace. 1.5 cc. alcohol mixture. | None. | Repeated twice with 2 cc. alcohol mixture. | None. | No relief. | None. | Failure. Patient had a large sacral cyst which may have communicated with the subarachnoid spaces. |
| (11) | Carcinoma bladder. | Perineum, right leg, vesicovaginal fistula. Area supplied by $D_{12}$, $L_1$, $S_3$, $S_4$, $S_5$. | 1st lumbar interspace 2 cc. alcohol mixture. | $D_{12}$, $L_1$, $L_2$. | None. | | Only slight relief for seven days—gradual improvement—quite comfortable six months after injection. | Slight motor weakness. | Patient also had roentgen-ray therapy—difficult to determine actual cause of relief. |
| (12) | Carcinoma testicle. | Right leg. Area supplied by $S_3$, $S_4$, $S_5$. | 1st lumbar interspace. 2 cc. absolute ethyl alcohol. | $S_3$, $S_4$, $S_5$, $S_1$, $S_2$, $L_4$, $L_5$, $L_6$, $L_4$, $L_5$. | None. | | Complete relief of pain for eight days (death). | Motor weakness. Retention of urine. | Excellent except for complication. |
| (13) | Carcinoma bladder. | (1) Left leg—area supplied by $L_4$, $L_5$, $L_6$, $L_6$, $L_6$.<br>(2) Recurrence in right leg—$L_4$, $L_5$, $L_6$, $L_6$.<br>(3) Recurrence on left leg—$L_4$, $L_5$, $L_6$, $L_6$.<br>(4) Persistent pain in right leg—$L_4$, $L_5$, $L_6$, $L_6$. | (1) 1st lumbar interspace. 1.5 cc. alcohol mixture. | $L_4$, $L_5$, $L_6$, $L_6$. | (2) 12th dorsal interspace. 2 cc. ethyl alcohol.<br>(3) 12th dorsal interspace. 2 cc. ethyl alcohol.<br>(4) $L_4$, right. 2 cc. ethyl alcohol. | (2) $L_5$, $L_6$ involved partially.<br>(3) $L_4$, $L_5$, $L_6$, $L_6$, $L_6$, $S_1$, $S_2$, $S_3$.<br>(4) $D_{10}$, $D_{12}$, $L_4$, $L_5$, $L_6$, $L_6$, $S_1$, $S_2$, $S_4$. | (1) Excellent relief for three weeks, recurred within a few days.<br>(2) No relief.<br>(3) Very good relief.<br>(4) Good immediate relief—but pain recurred within a few days. | (1) None.<br>(2) None.<br>(3) Slight motor weakness.<br>(4) Marked motor weakness. | The first injection gave excellent relief for three weeks, but the results of the later injections were far from satisfactory. |

methyl alcohol, because in our experience it permits the use of amounts up to two cubic centimeters without complications. No matter how careful the technic, there will be cases in which reinjection is imperative even in unilateral involvement.

less the condition of the patient the more radical one can be in regard to dosage. The obscurity of the localization of the action of the injected alcohol, and the potential danger involved, preclude its use in benign conditions. For patients having incurable carcinoma the

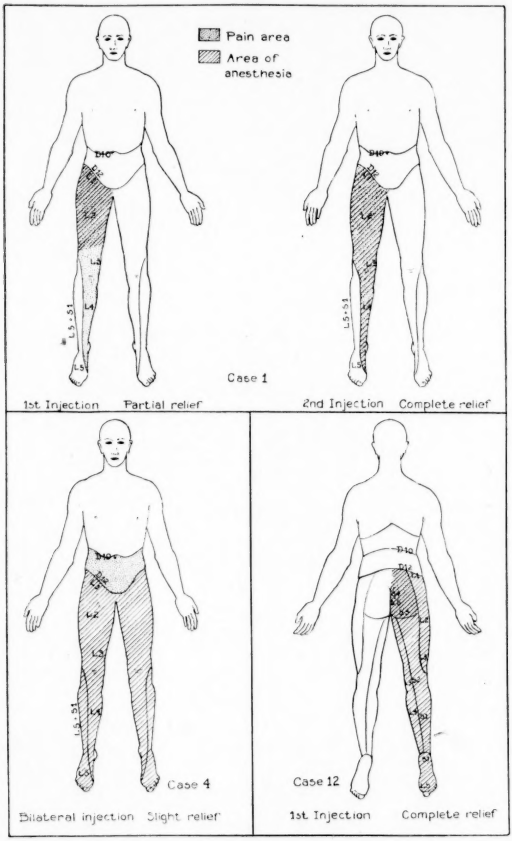


FIG. 5. In successful cases the anesthetic areas and painful areas as mapped out on the skin have coincided.

It is evident that while the subarachnoid injection of alcohol for the relief of intractable pain is very useful, it has not always been satisfactory. Moreover, the margin of safety between sensory relief and motor damage is small. Consequently, we feel that for the present at least, this procedure should be reserved for patients with incurable disease. The more hopeless procedure can be recommended without reservation.

## SUMMARY

1. Results are reported of observations on thirteen subarachnoid injections of alcohol for the relief of pain.

2. Excellent results were obtained in six

cases; incomplete relief in five cases; failure in two cases.

3. In those cases in which complete relief was not obtained the nerves to the painful area were not adequately blocked, as the anesthetic areas and painful areas did not coincide. Visceral pain is less amenable to this form of therapy than is somatic pain.

4. The method is recommended for the relief of intractable pain in patients with advanced cancer.

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# PRIMARY CARCINOMA OF THE JEJUNUM WITH REPORT OF TWO CASES

BY E. M. HODGKINS, M.D.*

PRIMARY adenocarcinoma of the small intestine, whatever the location, is so rare in the individual experience that most authors on the subject consider it a pathological curiosity. Recent reports emphasize the fact that this is especially true of the proximal jejunum. A review of the literature on the general subject of small intestinal malignancy immediately impresses one with the absence of a satisfactory working hypothesis for dealing with the lesion. This fact is not surprising when one considers the small number of cases thus far reported. Evidently no single surgeon or group of surgeons has encountered all of the varied difficulties in diagnosis and treatment that may arise from the many forms of this disease and therefore definite conclusions are not justified. Collective grouping of the cases has been most unpromising. In spite of this, the effort toward a better understanding of the subject goes on within a group of large clinics which will eventually dispel present confusion.

From a diagnostic viewpoint; sex and age incidence, early and late symptom syndrome, roentgenologic characteristics, predilection for location, cell morphology, period of metastatic activity in different types, all of which are so important in other cancer locations, fail to find agreement here. The following brief statistical abstracts from the literature are interesting and will serve to emphasize the rarity of the disease and certain striking disagreements.

The necropsy tables afford the best statistical account of small intestinal cancer and the reports of Nothnagel and Geiser are impressive. The former found only ten cancers of the small intestine in a total of 3,585 cancers in 41,838 necropsies; Geiser but two in a total of 909 cancers in 11,314 necropsies.

Muller, in 5621 necropsies, found cancer of the jejunum and ileum in only three cases. Proportional number in each not stated.

Forgue and Chavin in a series of 88,031 necropsies found carcinoma of the small intestine in only .04 per cent of the total examinations.

Bunting reported one case of carcinoma of the small intestine in 2,200 necropsies.

McKenty reported two cases occurring in the small intestine in 2,500 necropsies.

Johnson, at the Vienna General Hospital, in 41,838 necropsies, found 343 intestinal cancers ten of which were in the ileum and none in the jejunum.

Clinical reports show great variance in incidence. For example: In 1919 Judd reported twenty-four personal cases of primary cancer of the small intestine, eleven of which were located in the jejunum.

Craig, in 1924, studying the regional lymph glands in thirty-six cases of small intestinal cancer found only four cases of primary carcinoma of the jejunum.

Vickers, in 1924, stated that for any given unit of length of small gut, the jejunum shows the greatest relative immunity to cancer. Allbutt and Rolleston have noted that in their experience primary carcinoma of the jejunum is very rare.

Morrison, in 1927, writing on primary carcinoma of the proximal jejunum, stated that the lesion is very uncommon and is usually discovered at autopsy or when the abdomen is opened for other causes.

Bengolea and Baztterrica in 1930, writing on cancer of the small intestine, stated that the most frequent location of primary cancer is the terminal ileum, and except for the duodenojejunal junction, primary carcinoma of the jejunum does not exist. This is a most contradictory opinion.

Newton and Buckley, in 1930, emphasized the rarity of this lesion by summing up the re-

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ported statistics of European clinics and adding personal reports from eight of the largest hospitals in this country. The result was thirty-five histologically verified cases among 135,000 necropsies.

Bland-Sutton, in 1914, was of the opinion that carcinoma in the duodenum occurred more commonly than in either the jejunum or ileum.

Rankin and Mayo in 1930, writing on carcinoma of the small bowel, stated that in the Mayo Clinic up to that time, there had been only fifty-five cases of carcinoma of the small intestine as against 4,597 of the large bowel including the rectum. In twenty-one cases (38 per cent) the carcinomata were found in the jejunum. They also found that a surprising number of these were at, or a short distance from, the ligament of Treitz. These authors excluded cases of small intestinal carcinoma found in combination with carcinoma elsewhere.

## CLINICAL PATHOLOGY

Briefly, there are two general types of carcinoma of the small intestine, i.e., the adenocarcinomas and the carcinoids. The former, although in themselves rare, are well understood pathologically and comprise the larger group of epithelial tumors occurring in this location. The clinical gross appearance is that of an annular constricting tumor partially or completely obstructing the lumen of the bowel, not greatly dissimilar from that of the large bowel tumor. Metastasis takes place early to the adjacent lymph nodes of the mesentery. While infrequent, adenocarcinoma does occur anywhere throughout the entire length of the small intestine. Nevertheless it seems to have a predilection for the first portion of the jejunum near the ligament of Treitz (eighteen to twenty inches distal). From this segment downward, the rest of the jejunum and of the ileum are curiously spared. The terminal ileum is rarely involved at or near its union with the cecum (ileo-cecal valve) but involvement several inches above is relatively frequent. Judd states that in his cases of carcinoma of the ileum, nearly all the growths were several inches above the valve. Most surgeons and pathologists who have had the opportunity of examining malignancy at or directly involving the valve find evidence to indicate that the source of origin is on the large bowel side, and extension by continuity involves the terminal ileum.

In a study of fifty-seven cases Soper found that adenocarcinoma showed a predilection for that part of the small intestine nearest the stomach and colon respectively and an analysis of cases reported by others seems to lend support to Soper's findings.

In accord with this viewpoint, Johnson suggests that the fluid nature of the contents, the alkalinity of the intestinal fluid and the absence of abrupt bends in the small intestine may be the important factors in explaining this infrequency of growths. There are, however, those that believe that the lesions in the jejunum exclusively are either all metastatic from parent tumors elsewhere or possibly islands of aberrant tissue which have undergone malignant change. Secondary malignant degeneration of aberrant tissue is a proved pathological entity for other locations in the body and therefore is logical here at least in theory if not in fact.

Seidelin and Hernandez have both reported cases of aberrant pancreas in the jejunum which had undergone malignant degeneration and believe that this should always be considered among the causes of carcinoma at this location. Ransom, Oberndorfer, Verse, and Schopper have described what they prefer to call carcinoid tumors of the small intestine as distinguished from adenocarcinoma.

The pathology of carcinoid tumors at present is not well understood so that their ultimate development must still remain in doubt. A large number probably atrophy and disappear entirely. Some may remain stationary and never produce symptoms while a small group will undergo malignant degeneration.

They are variously described as originating from embryonic rests, from the cells of Lieberkühn's crypts, from aberrant pancreatic tissue, or from basal cell elements in the submucosa. It is, however, now realized that an indeterminate percentage *will* show pancreatic tissue.

In contradistinction to adenocarcinoma, carcinoids that go on to proliferation are said to extend by local invasion, growing to large size very slowly, often obstructing without metastasizing at all or starting metastasis very late. The consensus is that they are practically confined to the jejunum.

## SUGGESTED TREATMENT

From analyzing a large number of reports on the subject of carcinoma of the small intestine a practical working hypothesis is suggested. Disregarding the duodenum it may be assumed that the preponderance of malignant epithelial tumors occur at the *near* proximal jejunum and the *near* terminal ileum. Thus, the exploring surgeon, in searching for suspected malignancy in the small intestine is able immediately to eliminate these areas and by so doing save valuable time and handling of the bowel. In general, the treatment of large malignant bowel tumors is complete removal of the area involved with its adjacent mesentery. In the instance of small carcinoid tumors discovered early, wide removal locally without resection of the adjacent mesentery should be sufficient to effect a cure. Large carcinoids either partially or com-

pletely obstructing the bowel should be treated as potential adenocarcinomas. End-to-end or end-to-side anastomosis reëstablishes the continuity of the bowel after resection.

In the presence of metastasis or because of the attendant obstruction when one does not feel justified in resecting the bowel, a palliative entero-anastomosis may be used to sidetrack the pathological lesion thereby relieving the obstruction and prolonging life.

In support of the theory that aberrant pancreatic tissue produces carcinoid tumors in the jejunum and does undergo malignant degeneration, the following illustrative case is submitted. In addition, a case of primary adenocarcinoma of the jejunum in which early operation resulted in cure is reported.

## CASE REPORTS

CASE 1. Mr. L. J., white, aged sixty-four years, was seen in consultation with Dr. A. N. Allen, June 2, 1927. The patient gave a history of periodic attacks of indigestion over a period of twenty years. All food eaten produced distress in the region of the epigastrium. "Heartburn" came on immediately after eating followed by a feeling of nausea. He vomited only occasionally and there was no blood in the vomitus at any time. The vomitus contained sour tasting, undigested food. The patient never noticed any tarry appearance of the stools.

In 1926, he had a severe attack of pain in the abdomen with continuous vomiting of everything ingested. Under his physician's care, he was given medicine and placed on a liquid diet. Recovery was prompt. Roentgenological examination was suggested to him but this was deferred inasmuch as he felt so much better. He continued on a restricted diet, with reduction of symptoms. More recently symptoms have been getting worse with inability to take even liquids without pain and fullness in the epigastrium and frequent vomiting. Nausea is now present when the stomach is empty which has never been experienced previously. There is still no blood in the vomitus. He feels weak and tired constantly.

*Physical Examination:* The patient was fairly well developed, although poorly nourished. His skin was dry and showed no jaundice. The findings of the heart and lungs were within the normal. Reflexes were normal.

The abdomen was scaphoid in appearance, the skin over the abdomen was thin and loose and almost devoid of subcutaneous fat. Weight 107 pounds. Abdominal palpation revealed general mild tenderness throughout with a specific sensitive point with spasm high in the epigastrium just slightly to the right of the mid-line. There was thought to be a possible mass on deep pressure but this was not certain.

*Laboratory Examinations:* The laboratory examinations showed the white blood count to be 9,700. Red blood count 4,000,000. Urine concentrated although otherwise normal. Stools showed faint blood by guaiac test. A test meal for analysis of the stomach contents was not done.

*Roentgenological Examination:* Barium meal was given. The greater and lesser curvatures of the stomach were well defined. The duodenum filled incompletely. Six-hour examination shows a two-thirds stasis of one-half the meal.

Roentgenological Diagnosis: Obstruction of the pylorus, possible ulcer. Malignancy is a strong possibility in this case.

*Diagnosis:* Pyloric obstruction, malignant.

*Operation:* Under nitrous oxide gas, oxygen and ether, the abdomen was opened through a high right rectus incision. Exploration showed the gallbladder, pancreas, kidneys and spleen to be normal. Examination of the stomach revealed an almost complete obstruction at the pylorus due to multiple ulcers and wide induration. The duodenum was normal. Resection of the pylorus and ampulla of the stomach was decided upon. The ampulla of the stomach was adherent to the pancreas, but separation was done without causing injury to this structure, or excessive hemorrhage. The cut ends of the stomach and duodenum were closed tightly in the usual manner as a Billroth No. 2 type of anastomosis seemed advisable. On drawing up the proximal loop of jejunum, in preparation for posterior gastrojejunostomy, a small tumor was discovered on the anterior wall about eleven inches from the ligament of Treitz. This tumor was approximately the size of a five-cent piece, ovoid in shape, hard, discrete with excrescences on the surface and yellowish gray in appearance. The appearance was that of an early carcinomatous tissue. This presented a problem as the tumor was located at just the point where anastomosis should be done. Local excision was possible as the jejunum was of good size and the loss of tissue would not interfere with a good anastomosis. Clamps were applied around this area and the tumor removed by means of an elliptical incision. The resulting aperture was anastomosed to the posterior wall of the stomach and made a satisfactory stoma. The patient stood the operation well, leaving the operating room with pulse of 114, good volume. Treatment was administered in bed for mild shock.

*Pathological Report:* Specimen of ulcer of jejunum received June 3, 1927.

Microscopic examination shows adenocarcinoma. A considerable amount of pancreatic tissue was present in the muscularis.

Specimen from pylorus received June 3, 1927.

Microscopic examination shows no malignancy. Specimen from gland shows no malignancy.

*Outcome:* This patient has been seen frequently in the past eight years by Dr. Allen and the author. He remains in excellent health and his present weight is 140 pounds.

The following roentgenological examination by Dr. Albert M. Moloney, February 27, 1932, is interesting in that it shows a satisfactorily functioning stomach and jejunum.

*Roentgenological Examination by Dr. Albert M. Moloney:* Check-up films show a smooth-bordered, operative defect in the distal part of the stomach, resulting from resection of its pyloric end. The stomach appears otherwise negative, except for a gastro-enterostomy stoma that allowed media to flow out of it.

By fluoroscopy, peristaltic waves were seen to pass symmetrically along the course of the stomach. There was no tenderness or apparent defect or intraluminal abnormality noted at the site of defect. On the dependent part of the greater curvature of the stomach, the gastro-enterostomy stoma was found to be functioning normally and there was no tenderness over it.

Films taken one hour after ingestion of the barium meal showed the media flowing freely through normally appearing loops of jejunum in the upper abdomen.

CASE 2. Mr. J. C., aged fifty-one years. April 4, 1930, this patient came to the clinic complaining of what he termed "pressure" in the abdomen. Sharp pains and cramp-like sensations came on some-

times about two or three hours after eating accompanied by transient nausea and a feeling of weakness. At the onset, which was about a year previous, he did not consider it serious enough to consult a physician because certain patent medicines seemed to give relief. He was only mildly constipated at the beginning, but now goes two or three days without moving the bowels. He has not noticed blood in the stools. One attack of pain was very severe, accompanied by vomiting. There has been no blood in the vomitus, however. Pain, at this time, had radiated around the umbilicus and there was a feeling of increased pressure in the epigastrium. Other than the above complaints, the patient felt well and continued his carpentry. His appetite was fair, and he did not think that he had lost any weight although he couldn't be sure.

*Physical Examination:* The patient was well developed and nourished for a man of fifty-one years. The head, neck and chest were normal with the exception of some gingivitis of the upper and lower incisors. Heart and lungs were normal. Reflexes were carefully tested and proved to be normal.

The abdomen was moderately fat, undistended, soft and nonspastic. At a point in the mid-line just above the umbilicus there was some tenderness on deep pressure, although not remarkable or accompanied by muscle spasm. No mass was felt.

*Laboratory Examinations:* Wassermann was negative. White blood count 9000. Red blood count 3,400,000. Stools negative for blood. Urine negative. Gastric test meal showed total acidity normal, free hydrochloric acid content normal, no blood, Oppler-Boas bacilli or sarcinae.

*Roentgenological Examination:* Gastrointestinal: No organic abnormality was found in the stomach. It is of normal size and shape and in six hours is entirely empty, showing good motility. The duodenum fills fairly well, uniformly and reveals no characteristic signs of ulcer.

In twenty-four hours the large bowel was empty. At this time we gave a barium enema which revealed the large bowel to be uniformly filled from the rectum to the cecum. There is no evidence of stenosis or in fact any abnormal changes. There is nothing to suggest an organic lesion.

Films of the gallbladder with the Graham test show it to be normal.

*Tentative Diagnosis:* Intestinal malignancy. Location undetermined.

*Operation:* April 8, 1930. The abdomen was opened through a mid-epigastric incision and exploration was done. Stomach, pylorus, duodenum, gallbladder, pancreas and both kidneys were examined and found to be normal. The incision was enlarged and the appendix drawn up into it. The appendix was normal. In exploring further, a tumor mass was palpable in the omentum just to the left of the incision. With wide retraction and closer investigation, the tumor was found to be behind the omentum, rather than in the omentum, and located in the proximal jejunum about fourteen inches from the ligament of Treitz. The tumor was enveloping and constricting the bowel so as to cause partial obstruction. There was a good deal of edema both on the proximal and distal sides of the obstruction with dilatation of peritoneal surface vessels. Mesenteric lymph nodes were not enlarged and it was assumed that metastasis had not begun. Complete resection of the tumor and adjacent mesentery was decided upon as advisable and the operation proceeded without difficulty or untoward event. The bowel was anastomosed end to end. An enterostomy tube was considered unnecessary and therefore the abdomen was closed tightly.

This patient's first five days of convalescence were rather stormy, mild distention being the chief difficulty. After this period, the convalescence was smooth and he was discharged from the hospital on the sixteenth day.

*Pathological Report:* April 9, 1930. Received a specimen of jejunum with mesentery attached, and containing constricting tumor.

The section shows a hard mass completely enveloping the intestinal circumference, partially obstructing the lumen.

Microscopic section shows adenocarcinoma.

Pathological Diagnosis: Carcinoma of the jejunum.

*Outcome:* This patient has remained in good health for the past five years. He states that he has been entirely free of former distressing symptoms.

## COMMENT

To draw conclusions from the two cases herein reported is manifestly not the purpose of this paper. In Case 1, carcinoma of the proximal jejunum was discovered accidentally while resecting a portion of the stomach for obstructing ulcer. Case 2 was undiagnosed preoperatively on account of insignificant symptoms, negative roentgenologic examination and laboratory findings. Exploratory operation was resorted to on account of the patient's age and chronicity of symptoms, such as continued crampy abdominal pains, constipation, distention, transient nausea and moderate anemia, all of which are suggestive of cancer somewhere in the intestinal tract.

To reiterate: despite the rarity of carcinoma of the proximal jejunum and terminal ileum, the exploring surgeon will do well to include both these areas in his points of investigation when operating upon a patient, within the cancer age, for gastrointestinal disease.

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# CASE RECORDS
## *of the*
# MASSACHUSETTS GENERAL HOSPITAL

ANTE MORTEM AND POST MORTEM RECORDS AS USED IN WEEKLY CLINICAL-PATHOLOGIC EXERCISES

FOUNDED BY RICHARD C. CABOT, M.D.

TRACY B. MALLORY, M.D., *Editor*

## CASE 22101

### PRESENTATION OF CASE

A twenty-nine year old white native college student was admitted complaining of abdominal pain.

For several years the patient suffered from weekly attacks of nausea occasionally accompanied by vomiting. He felt generally well, however, and presented no further symptoms until one and a half years before entry, at which time he passed a stool which was said to have been black. On two occasions thereafter he passed similar stools. One year before admission he began to suffer from dull gnawing pain in the mid-abdomen. The pain usually occurred after his midday meal but frequently had no relationship to meals. It did not radiate nor were there any other associated symptoms except for occasional watery diarrhea, which recurred approximately once a month. There was gradual increase in the frequency of the pain until it appeared daily and ultimately two to three times daily. For about three months there was increasing fatigue, lethargy, and some malaise. Five weeks before entry he developed a severe sore throat which over a period of two weeks developed into a "head cold". At this time he consulted a physician who, after examining his blood and stools, advised hospitalization.

Physical examination showed a well-developed and nourished man in no distress. No positive physical findings were elicited. The heart and lungs were negative. The blood pressure was 120/65.

The temperature was 99°, the pulse 88. The respirations were 22.

Examination of the urine was negative. The blood showed a red cell count of 4,350,000, with a hemoglobin of 70 per cent. The white cell count was 8,300, 67 per cent polymorphonuclears. A proctoscopy was negative but feces removed at this time gave a positive reaction to the guaiac test as did two subsequent specimens.

A barium enema showed normal filling and emptying of the colon. There were no evidences of polypi. The shadow of the spleen appeared to be rather prominent. Several days later an examination of the esophagus, stomach and duodenum was negative. At six hours the motor meal had reached the hepatic flexure. There was a localized dilatation of the terminal ileum about one foot from the ileocecal valve. The dilated loop lay low in the true pelvis and gradually reduced in size proximally so that within a short distance it appeared to be of normal caliber. Peristaltic contractions of the dilated loop were vigorous but there was no persistent spasm.

On the fourth hospital day a laparotomy was performed.

### DIFFERENTIAL DIAGNOSIS

DR. BETH VINCENT: We have here a comparatively young man, twenty-nine, whose chief symptom is abdominal pain. He was sent to the hospital where examination of his stools showed occult blood. The history states that one and a half years before entry he may have passed tarry stools. So we have to deal with the diagnosis of a case of abdominal pain with bleeding from the intestinal tract. In all cases of bleeding I think it is well at first to look very quickly at the blood picture and rule out the possibility that you are dealing with a pathological hemorrhage such as we get in thrombopenic purpura. The blood examination given here enables us to exclude that and in passing we note we are not dealing with one of the primary blood diseases.

We also note in the physical examination that the examination made by proctoscopy enables us to exclude one of the most common causes of bleeding, hemorrhoids, and at the same time confirms the fact that the blood was in the stools and thus came from somewhere along the gastrointestinal tract. Such bleeding may indicate either a break in the continuity of the mucous membrane of the gastrointestinal tract, as in ulcer, or the ruptures of blood vessels some place in the gastrointestinal tract that have become unduly numerous or dilated. The last we might find in a case of angioma of the intestines, a rare lesion and so difficult of diagnosis I think we may dismiss it in this case. Another condition which predisposes to bleeding from ruptured vessels is more common and deserves further consideration; that is, where the veins of the stomach or esophagus are numerous and dilated on account of obstruction of return of the portal blood. This takes place in two diseases; first in atrophic cirrhosis where the obstruction of the venous return is within the bed of the liver; and secondly, in obliterative thrombophlebitis of the portal vein or its radicles where the venous return is obstructed in the portal or splenic veins. In both these instances, especially in an individual as old as this patient, there is usually congestive enlargement of the spleen. You will notice in this case that the

physical examination was negative and therefore the spleen cannot have been much enlarged, as usually it is easily felt. X-ray suggests some enlargement of the spleen, however. Also, the bleeding in these cases is usually massive. Here the history at one time suggests bloody stools, but there was no vomiting of large amounts of blood.

As to cirrhosis there is no ascites. The individual is rather young for cirrhosis and apparently the diagnosis was not very seriously considered because we have no determination of the functional capacity of the liver. So I think we are safe in saying that this diagnosis is rather improbable.

Continuing then with the ulcers of the gastrointestinal tract, if we begin with the stomach and duodenum we would have to consider malignancy of the stomach and peptic ulcer of the stomach or duodenum. The pain, the gastric symptoms, and the black stools might suggest a peptic ulcer, but you will note that the x-ray is negative. We know that failure to develop the lesion by x-ray in these two diseases, peptic ulcer of the duodenum or stomach, or malignant ulcer of the stomach, is rather rare, so I think perhaps we are justified in saying that this diagnosis is improbable. In speaking of peptic ulcer and bleeding we ought to consider a peptic ulcer in another situation; that is, in relation with a Meckel's diverticulum. As you know, we have those cases. They usually occur in infancy or childhood, may give pain, may give blood in the stools, with a negative x-ray. But most of these cases manifest themselves in infancy or childhood, so I should think this diagnosis was also unlikely.

Passing down into the intestinal tract, if we begin at the other end you will note that the rectum by proctoscopy was negative. I take it also that the digital examination was negative, which certainly would rule out most of the cases of malignancy of the lower bowel. I mean by that, from the rectosigmoid junction downward, and since the x-ray examination of the colon was negative and they did not feel any tumor, it seems to render it unlikely that there was malignancy of the colon. At the same time the x-ray examination of the colon is against ulcer due to colitis and the picture of the individual does not conform to that of ulcerative colitis. It is only fair to say, if this is a surgical lesion, and perhaps because I was asked to discuss it it may be, that most of the low surgical lesions that cause bleeding are located in the colon or terminal ileum. That brings us around to the terminal ileum or cecum. This man has a moderate secondary anemia although x-ray is negative and they felt no tumor. I suppose he could have a malignancy of the cecum, although we can scarcely account for the symptoms of a year and a half with that diagnosis. He has some temperature, and he might have an inflammatory lesion of the cecum such as we see in tuberculosis, or a lesion of the terminal ileum that is sometimes found, but often hard to diagnose; that is, regional ileitis.

Before making an attempt to settle on any exact diagnosis I think we need an expert's interpretation of the x-ray findings.

DR. AUBREY O. HAMPTON: I have so many films I am lost trying to pick the best ones. We knew this man was bleeding and we looked for a source. We did practically everything we could including a double contrast enema and a particular search for a posterior wall duodenal ulcer. The double contrast enema was a total washout because the barium did not stick to the mucosa. Here is the gas-filled colon and there is little barium in it. We were able to examine the colon though without any difficulty. There were no redundant loops and you could palpate the whole thing. I think we were quite convinced that there were no polyps. The only positive finding that we had was dilatation of the terminal ileum and this was quite definite. Here is the ileocecal valve, the terminal ileum coming over here, down and around and across and up like that. This loop is quite a way from the ileocecal valve, about two feet, and we were unable to displace it upward although the patient tolerated heavy deep pressure very well. I think it was fixed in that position, deep in the pelvis. One of the films we had showed a small pouch-like thing that was superimposed upon the abnormal bowel here which turned out later to be the tip of the appendix. Of course, that brought up the suggestion that was made, that it might be a Meckel's diverticulum. Certainly at the six-hour examination it was in the location of a Meckel's diverticulum. Later we found that the appendix was unusually low and long, superimposing on the ileum in the midline. But I do not think there was any doubt in any of our minds that the ileum was dilated at that point.

DR. VINCENT: I should think then we might hope to find the lesion in the right lower quadrant and I think perhaps we should exclude an inflammatory or malignant lesion of the cecum and should hope that we would find one of these cases of regional ileitis. If I were going to explore this man I certainly would make a right paramedian incision.

DR. HAMPTON: The x-ray findings were not those of regional ileitis.

DR. VINCENT: In spite of that, I would go over the terminal ileum and cecum and, although they report no polyps, would feel called upon to palpate the colon for polyps very carefully, although as a rule this examination is unsatisfactory. There may be polyps there which cannot be found. At the same time one should feel the liver because if there was a pronounced cir-

rhosis of the liver we could make that diagnosis. It may be said that in these cases of bleeding from the intestinal tract where the preoperative diagnosis is so uncertain the exploration is often negative. I do not believe it is so in this instance because otherwise Dr. Mallory would not have presented the case for our consideration.

DR. TRACY B. MALLORY: Have you any comment, Dr. Jones?

DR. CHESTER M. JONES: I know what he had, but there are certain points that I should like to make. I think it is interesting in the first place because this student reported to one of the men over at Harvard a year ago and said he was fatigued and on examination nothing was found. Fatigue was the outstanding symptom. He came back this time with the same symptom. At that time a blood was done and the hemoglobin was said to be seventy but it was seventy Sahli and whoever did it paid some attention to it. Seventy per cent Sahli in a young man of twenty-nine represents a definite but rather slight anemia and on the basis of that a careful study was made of this man in the office over at Cambridge as to the cause of his anemia. It was thought not to be a primary blood disease but secondary to loss of blood, and stools were taken and a positive guaiac found. I think that represents an intelligent attempt to answer this patient's symptoms in the first place. One of the things not brought out in this history is the fact that this boy's pain was usually merely discomfort but on one or two occasions was sharp, localized below the umbilicus in the midline, very strongly suggestive of small bowel pain as contrasted with that due to disease of the stomach, duodenum or large bowel. He persisted in localizing that pain always in the same place. I proctoscoped him and the examination was negative except that we did get fecal material coming down from above showing a positive guaiac test. That was important because it showed that there was bleeding from above. The x-rays were taken and Dr. Hampton discussed them with me. We felt because of the local ileal dilatation, the symptoms, and because of the bleeding that this might represent the very unusual picture of Meckel's diverticulum bleeding in an adult. It was on this basis that we explored, with a diagnosis of Meckel's diverticulum, and we expected to relieve it.

A PHYSICIAN: How often do they show by x-ray?

DR. HAMPTON: I thought I was going to be the first one in the hospital to make the diagnosis by x-ray and I thought right up to the second note that this was due to a Meckel's diverticulum, but since I could not demonstrate the diverticulum I did not think I had enough to make a diagnosis. It was with the aid of the clinical findings, that Dr. Jones mentioned, that together we were convinced it was a Meckel's diverticulum. At the second examination I hunted for the diverticulum particularly, and I could not find it. The diagnosis by x-ray has been reported once in the literature.

PREOPERATIVE DIAGNOSIS

Meckel's diverticulum.

DR. BETH VINCENT'S DIAGNOSES

Regional ileitis?

PATHOLOGIC DIAGNOSES

Meckel's diverticulum.
Chronic ulcer.

PATHOLOGIC DISCUSSION

DR. MALLORY: This is a photograph of the specimen which was removed. You can see this wide diverticulum projecting to the left of the resected section of ileum. At the base of the diverticulum, just at the junction with the small intestine, there is an area which is discolored and shallowly ulcerated. The specimen had aroused so much interest that it received the treatment which sometimes occurs to particularly valuable specimens. It was taken out of the routine to be photographed and shown at various clinics, and by the time we got it back it was no longer of any use for histologic purposes. We did manage to get a single block for microscopic examination which shows the area of ulceration but does not show any gastric mucosa. The section was taken from the immediate neighborhood of the ulcer and it is the rule in these cases that the ulcer is not found in the area where the gastrointestinal mucosa is present but in the immediately adjacent intestinal type of mucosa. So there may perfectly well have been gastric mucosa here even though we did not demonstrate it. On the other hand I rather doubt if that was the case, however, because the ulcer does not look like a peptic ulcer. A peptic ulcer has microscopically a very characteristic appearance. The surface is usually quite clean, almost free from leukocytes, and then there is invariably in the active ulcer a superficial zone of so-called fibrinoid necrosis which appears in ordinary sections as a broad red hyalin layer. I have never seen an active peptic ulcer in which this was not present and there is no suggestion of it here. So I am inclined to think that this ulcer was infectious and not peptic in nature. If that is the case it might explain why he has gone well into his late twenties before developing symptoms instead of having hemorrhage from his diverticulum in the first and second decades as is the common story.

DR. MCKITTRICK: How high above the cecum may a Meckel's diverticulum be found? How much of the terminal ileum ought one to explore?

DR. MALLORY: I am not able to answer that

definitely. They say that a yard is the common spot but I am quite sure it can go higher, how much higher, I do not know.

A PHYSICIAN: How much of a lumen did it have? Could you stick your finger in it or not?

DR. MALLORY: Yes, very easily. It is characteristic that they almost invariably have a wide lumen as compared with an appendix, very nearly as great as that of the small bowel.

DR. HAMPTON: Is the mucosa in the diverticulum the same as in the terminal ileum, does it show the same gross pattern?

DR. MALLORY: It varies a great deal from one diverticulum to another. It should be approximately the same as the ileum. In some cases, however, a very large proportion is made up of gastric epithelium.

A PHYSICIAN: What percentage of cases of Meckel's diverticulum contain gastric epithelium in your experience, Dr. Mallory?

DR. MALLORY: I cannot answer that. It was supposed to be a very rare phenomenon until about five years ago, when the attention of surgeons working particularly in children's hospitals was called to the fact and on checking back they found that a very large proportion of these cases showed it. I do not know of any systematic study of the symptomless Meckel's diverticula that are so commonly found at autopsy. One and a half per cent of individuals have a Meckel's diverticulum. I do not know if any one has checked them to see if they contain gastric mucosa.

## CASE 22102

### PRESENTATION OF CASE

A sixty year old white Canadian woman was admitted in a semicomatose state.

Eight weeks before entry she went to care for a sister who was dying of pulmonary tuberculosis. A week later she became ill with pain, primarily in the left chest, but later upon the right side also. She had at the same time chilly sensations and some fever, and a physician told her she had pneumonia. Early in the course of her illness there was slight jaundice, but this disappeared in a short time. Her appetite was poor, and there was marked nausea but no emesis. Her mind "wandered" a great deal during her illness. She continued to run a febrile course for two weeks, at which time her tongue was so dry that three hypodermoclyses were given. Subsequently edema appeared, first upon the arms and feet but gradually involving the entire body except the face. The swelling remained unchanged upon the trunk and lower extremities but varied in severity in the arms, first affecting one and then the other. She required daily catheterization from the onset of her illness.

She had had rheumatic fever at the age of twelve years.

Physical examination showed a well-developed woman lying upon her back, tossing her head from side to side and moaning. Her respirations were Cheyne-Stokes in character. The skin was loose, dry, and flabby. The tongue was dry, swollen, and brown. The upper sternum was quite prominent. The heart was slightly enlarged and the sounds though regular were of a rather poor quality. A third heart sound was audible at the left border of the sternum and a systolic murmur was heard at the apex. Moist râles were present in the left lower back but no dullness was elicited. The abdomen was slightly distended and tympanitic. There was slight tenderness in the epigastrium with a vague sensation of an underlying mass. Marked edema of the right arm, both lower extremities, and the trunk was observed. Small decubitus ulcers were noted over the sacrum and right heel. The knee jerks were sluggish and the ankle jerks were not elicited.

The temperature was 99°, the pulse 100. The respirations were 32.

Examination of the urine showed a specific gravity of 1.006 with a trace of albumin. The sediment contained many white blood cells and an occasional red blood cell. The blood showed a red cell count of 3,340,000, with a hemoglobin of 75 per cent. The white cell count was 26,600, 91 per cent polymorphonuclears. The serum protein was 4.3 milligrams per cent. The nonprotein nitrogen of the blood was 53 milligrams per cent and the sugar 91 milligrams per cent. A lumbar puncture showed an initial pressure of 230 with the head slightly elevated. The fluid was clear, colorless, and contained 73 red blood cells but no white blood cells. The total protein was 57 milligrams per cent and the ammonium sulphate test was positive. A Hinton test was negative.

X-ray examination showed partial obliteration of the costophrenic angles. There were bands of dullness along each axillary line from the apex to the diaphragm. These bands were thicker in the region of the right interlobar septum and upper portion of the left lung. The lung fields showed diminished radiance as the result of a rather coarse, hazy mottling. The mottling was most dense in the region of the right upper lobe and interlobar septum between the upper and middle lobes. The right lung seemed smaller than the left and the intercostal spaces were narrowed on this side. The heart and mediastinum were slightly displaced to the right.

On the day following admission her temperature rose to 101°, but there was no change in her general condition. Despite the administration of transfusions she went rapidly downhill and expired on the third hospital day.

## Differential Diagnosis

Dr. Myles P. Baker: The gist of the problem here lies in the question of etiology of what on admission must have seemed a case with the so-called "slow nervous fever" of the old clinicians. The history in such a situation must of necessity be scanty in detail and only partly reliable, and so sick was she that there was no time for confirmation of certain laboratory examinations the results of which would warrant repetition.

We are dealing with a woman of sixty who has had a fever for at least two weeks, and probably seven, who is brought to the hospital in a restless delirium with signs of dehydration and edema of the trunk and extremities, but not of the face, the latter so typical of renal edema. As for the details of her illness, we do know that the infection began with pain first in the left chest and later on the right side, presumably pleural pain. Of the importance of the slight jaundice mentioned we cannot judge and it is probably hazardous to emphasize this.

It is interesting that from the very beginning she was confused mentally and required daily catheterization for incontinence in all likelihood.

The appearance of edema varying in severity in the arms according to which side she lay on bespeaks a plasma protein deficiency.

It seems fair to wonder whether this fatal infection did not follow on a period of relative ill health with impaired appetite and protein intake, but such must be only conjecture.

The physical examination is noteworthy in that there is no localization of the infection other than such evidence as there is of possible pulmonary infection in the left lower lobe. The absence of ankle jerks is not to be wondered at in such a depleted individual. We have no other evidence of a peripheral neuritis.

The increased respiratory rate is worthy of comment and directs attention toward the possibility of a more widespread pulmonary involvement than one would conclude from the physical examination.

Laboratory tests reveal the picture of a cystitis. This may well be the result of repeated catheterization. She was anemic. One would hesitate to draw any conclusion from one hemoglobin determination of seventy-five per cent with such a low red blood cell count. The leucocytosis is important. The serum protein level is what we would expect with the degree of edema noted and seven weeks of fever and neglect. The nonprotein nitrogen we may interpret as evidence of dehydration rather than renal insufficiency necessarily. It is not the urinary sediment of a progressive glomerulonephritis, nor have we had the retinopathy of such. The spinal fluid shows findings that are difficult to interpret. The slightly elevated pressure and rise in protein are compatible with the presence of an area of degeneration adjacent to the subarachnoid space in an elderly person with tiny cerebral thromboses. It is strange that she should have as many red blood cells as this without any white blood cells. An increased spinal fluid protein is occasionally found in elderly people with cerebral arteriosclerosis and the main information to be drawn from this wisely done lumbar puncture is that the woman did not have a subarachnoid hemorrhage, which is sometimes the cause of a "typhoidal" state, and protracted fever.

Prior to the evidence brought to hand by the chest x-ray several possibilities might come to mind, among them miliary tuberculosis; a protracted case, with recrudescences, of typhoid fever which had begun with an initial bronchitis, or gone on to some complication such as a pyelonephritis with colon bacillus infection. Were this a neglected undiagnosed case of typhoid fever there might have been a crural thrombosis that had suppurated or suppurating mesenteric glands that had given rise to a leucocytosis. It is well to remember that miliary tuberculosis and typhoid fever may occur together, or that pulmonary tuberculosis may manifest itself in the course of convalescence from typhoid fever, and the fact that typhoid fever begins with pleuritic symptoms, simulating the effect of rupture of a caseous subpleural lymph node into the pleural cavity, is of course an old story.

The fact that she had had rheumatic fever early in life makes one consider the possibility of a chronic ulcerative endocarditis only to abandon the possibility, for there have been no embolic phenomena for one thing.

Some cases there have been of obscure, deep seated osteomyelitis undiagnosed until postmortem, but without localizing signs this must have seemed unlikely.

I recall seeing one case of delirium with similar fever and dehydrated state, a woman who showed on physical examination only the signs of an acute bronchitis. This was the first of the cases of tularemia with pneumonia recently reported by Bernstein in the Hopkins Bulletin.

Her chest x-ray, as I recall it, revealed nothing of a localizing nature. This brings us to a discussion of the x-ray of this patient's lungs which seems to me to limit the diagnostic possibilities. The x-ray picture showing hazy mottling, most dense in the region of the right upper lobe but involving both lungs, is consistent with the presence of a miliary tuberculous infection. I gather that the shadows are not so sharply demarcated and dense as in the accidentally found healed stage of miliary tuberculosis, nor are the shadows those of the larger nodules, more round, less uniform, that are char-

acteristic of carcinomatosis of a miliary type. It is not the picture of lymphangitis carcinomatosa rarely seen in association with a scirrhous carcinoma of the stomach which has metastasized to perivascular lymph channels in the lungs. There is not the predilection for lower lobe involvement of silicosis. The smaller right lung field is I suppose due to partial bronchial occlusion, as with an older peribronchial fibrosis or a newer necrosis of the mucous membrane. The x-ray, in the absence of evidence of carcinoma elsewhere, seems to me to point strongly to the diagnosis of miliary tuberculosis which is a self-propagating septicemia occasionally running as long a course as this, seven to eight weeks. The onset with pleuritic pain, the typhoidal course, the rapid respiratory rate, the leucocytosis, all are consistent with such a diagnosis. It is interesting to note that in the earlier Cabot Case Histories miliary tuberculosis was often missed in the antemortem diagnosis; but the x-ray examination here is an added aid in the diagnosis and confirms my suspicion that it is of a miliary tuberculosis that this woman died.

### Clinical Diagnoses

Acute and chronic nephritis with edema.
Unresolved pneumonia.
(? Miliary tuberculosis.)
? Brain abscess.
Secondary anemia.
Malnutrition.

### Dr. Myles Baker's Diagnoses

Miliary tuberculosis.
Cystitis.
Cerebral thrombosis.

### Anatomic Diagnoses

Organized thrombo-endarteritis of the pulmonary arteries.
Pulmonary fibrosis.
Emphysema.
Pyelonephritis, bilateral.
Cystitis.
Chronic pancreatitis with fat necrosis.
Hydrothorax, right.
Pleuritis, chronic fibrous, bilateral.
Ascites, slight.
Endocarditis, terminal, mitral.
Arteriosclerosis: Aortic, moderate.
Decubitus ulcers, sacrum and heel.
Perisplenitis.

### Pathologic Discussion

Dr. Tracy B. Mallory: This is a type of case which it is hardly fair to put up to the clinician for differential diagnosis inasmuch as when the autopsy has been done and all the microscopic slides carefully studied the pathologist is still unable to make a diagnosis. It belongs in a group of pulmonary cases which for the moment we are calling, simply because a name is necessary for filing, pulmonary fibrosis. It is obvious that this is not a disease but a condition of the lungs which can be produced by many diseases. The healed stage of a diffuse tuberculosis would, for instance, produce such a result. Organizing pneumonias not infrequently are another cause. Fibrosis developing in and about the lymphatics, such as we see in silicosis, represents another type.

This particular case belongs to a still different category. There are numerous patches of dense fibrous tissue in which the remnants of the elastica of the alveolar walls can still be seen. The picture is closely similar to that produced by organizing pneumonia. There is, however, another lesion present which I believe is primary rather than secondary. Nearly all the medium-sized and small pulmonary arteries show extensive organized and recanalized thrombi within their lumina. These lesions are for the most part old and inactive. A few lobules of lung tissue show fresh changes more suggestive of infarction than of pneumonia, and, in some of these, early organization is evident. I believe this case probably belongs to the group of diffuse thrombo-endarteritis of the pulmonary arteries. The possibility of multiple small emboli is difficult to exclude absolutely but no source for emboli was discovered in the remainder of the autopsy. The process is evidently one of great chronicity, of much longer duration than the patient's story would indicate. The lungs show the usual results of extensive pulmonary destruction in the form of marked emphysematous dilatation of the persisting alveoli. The heart weight was within normal limits, but the right ventricle measured 6 millimeters, a slight but definite cor pulmonale.

The clinicians in the hospital did not have a much more definite idea of the diagnosis than Dr. Baker. They had the advantage of personal consultation with the roentgenologist, who stated definitely that he did not believe the lesions were tuberculous although he could not rule out that possibility. The patient was discharged with a death certificate reading: ? unresolved pneumonia ? miliary tuberculosis. They believed that she probably had as well an acute and chronic nephritis with edema.

We found at autopsy slight injection of the renal pelves and slight pallor and thickening of the cortex. Microscopic examination showed a moderate grade of diffuse pyelonephritis. A few minute fresh vegetations were found upon the mitral valve. We considered this, however, only a terminal incident probably of little importance in the course of her disease. We unfortunately were unable to examine the head.

treatment of pulmonary tuberculosis as it develops at different ages. This discussion is to be of the sort which will be of value to the general practitioner rather than to those who are doing special work in tuberculosis. The papers are to be arranged as follows:

(1) Presentation of a case history of pulmonary tuberculosis in both infant and child. With discussion of treatment. Dr. Clement A. Smith, Boston Children's Hospital.

(2) Presentation of a case history of pulmonary tuberculosis in an adolescent. With discussion of treatment. Dr. Roy Morgan, Superintendent of the Westfield State Sanatorium.

(3) Presentation of a case history of pulmonary tuberculosis in an adult. With discussion of treatment. Dr. John B. Hawes, 2nd, Boston.

Dr. Edward D. Churchill, Professor of Surgery at the Harvard Medical School, will be present to discuss the cases from the surgical standpoint, and Dr. Hugh Hare, Roentgenologist at the Middlesex County Sanatorium, will be ready to discuss various features of the x-ray films.

The formal presentation of each case will be limited to twenty minutes so that there will be ample time for questions and discussion from the floor, and it is the hope of the officers that this part of the program will be of especial value. For safety's sake each discusser will be limited to three minutes.

A meeting of this sort is somewhat of an innovation for the annual session of the State Society, and it will be interesting to see whether such a program can be successfully carried through.

## THIS WEEK'S ISSUE

CONTAINS articles by the following named authors:

DALAND, ERNEST M. A.B., M.D. Harvard University Medical School 1918. F.A.C.S. Instructor in Surgery, Harvard University Medical School. Chief of Staff, Pondville Hospital, Wrentham. Assistant Surgeon, Massachusetts General Hospital. Address: 483 Beacon Street, Boston, Mass. Associated with him is

WELCH, CLAUDE E. A.B., M.A., M.D. Harvard University Medical School 1932. Formerly, Surgical Resident, Pondville Hospital. Now, Assistant Surgical Resident, Massachusetts General Hospital. Address: Massachusetts General Hospital, Boston, Mass. And

NATHANSON, IRA T. B.S., M.S., M.D. Northwestern University Medical School 1930. Littauer Research Fellow in Surgery, Harvard University Medical School. Formerly, Surgical Resident, Pondville Hospital, Wrentham. Address: Huntington Memorial Hospital, 695 Huntington Avenue, Boston, Mass. Their subject is One Hundred Untreated Cancers of the Rectum. Page 451.

SHATTUCK, GEORGE CHEEVER. A.B., A.M., M.D. Harvard University Medical School 1905. Assistant Professor of Tropical Medicine, Harvard University Medical School. Assistant Visiting Physician, Boston City Hospital, in charge of Service for Tropical Diseases. Address: Harvard University Medical School, Boston, Mass. Associated with him is

HILFERTY, MARGARET M. A.B., Ed.M. Formerly Statistical Technician in the Department of Vital Statistics, Harvard School of Public Health. Address: View Street, Leominster, Mass. Their subject is Distribution of Acute Heat Effects in Various Parts of the World. Page 458.

EVERSOLE, URBAN H. A.B., M.D. University of Kansas School of Medicine 1932. Anesthetist, Lahey Clinic, New England Deaconess Hospital and New England Baptist Hospital, Boston, Mass. His subject is Anesthetic Emergencies. Page 468. Address: 605 Commonwealth Avenue, Boston, Mass.

DUNPHY, J. E. M.D. Harvard University Medical School 1933. Assistant Resident Surgeon, Peter Bent Brigham Hospital. Address: Peter Bent Brigham Hospital, Boston, Mass. Associated with him is

ALT, R. E. M.D. Harvard University Medical School 1931. Formerly, Fellow in Urology, Peter Bent Brigham Hospital. Now, Resident Surgeon, Beverly Hospital, Beverly, Mass. Address: Beverly Hospital, Beverly, Mass. Their subject is The Relief of Pain by the Subarachnoid Injection of Alcohol. Page 472.

HODGKINS, E. M. M.D. Tufts College Medical School 1915. F.A.C.S. Assistant Professor of Surgery, Tufts College Medical School. Assistant Surgeon, East Wing, St. Elizabeth's Hospital. Surgeon-in-Chief, Roxbury Hospital. His subject is Primary Carcinoma of the Jejunum with Report of Two Cases. Page 477. Address: 45 Bay State Road, Boston, Mass.

## The Massachusetts Medical Society

### SECTION OF OBSTETRICS AND GYNECOLOGY*

C. J. KICKHAM, M.D., *Chairman*, 524 Commonwealth Ave., Boston, Mass.
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#### PREECLAMPSIA AND ECLAMPSIA

*Terminology and Definition:* The term "Toxemia of Pregnancy" should be abandoned

*A series of short selected articles by members of the Section is being published weekly.
Comments and questions by subscribers are solicited and will be discussed by members of the Section.

in favor of Preeclampsia and Eclampsia. These are the same condition except in degree.

Twenty-five per cent of all maternal mortality in the United States is accounted for by this condition.

*Etiology:* Hofbauer (*Am. J. Obst. & Gynec.* **26:** 311, 1933) showed to his satisfaction that the relationships between foreign protein (i.e., foreign to the body economy except in pregnancy) and the internal secretions, especially the posterior pituitary, result in preeclampsia and eclampsia. It is generally considered that certain steps in his reasoning remain to be proved or checked. Acceptance of his theory of etiology varies greatly among prominent workers in this field. It would perhaps be fair to state that many of the data involved are impressive, but that since certain links in the chain are weak his thesis is not in general considered proved. George van S. Smith seems to show a relationship between the pituitary and the eclamptic state, and quite likely it is along the endocrine line that a cause will be found. Considering the difficulty of concentrating the endocrines, the expense, and the large dosage presumably required, direct endocrine therapy seems a distant possibility, even if an endocrine relationship shall have been proved. For the present then we must fall back on the old idea; namely that the only sure known etiology of the eclamptic state is pregnancy, present or recently past.

It is now apparent that the group of cardiovascular and nephritic conditions which may be called, for the sake of simplicity, essential hypertension and suspected chronic glomerulonephritis are best viewed in relation to the eclamptic state from the standpoint of secondary etiological factors. In 1927 Corwin and Herrick (*Am. J. Obst. & Gynec.* **13:** 617, 1927) (*J. A. M. A.* **88:** 457, 1927) suggested this and in 1935 proved it by necropsy findings checked against a long period of clinical observation (*Arch. Int. Med.* **55:** 643, 1935). The exact and percentage relationship between these conditions and the eclamptic state has yet to be worked out in a great group of cases followed to autopsy; but a more or less general acceptance of this point of view is already apparent. As a basis for an up-to-date understanding of these medical conditions we have found the book "Hypertension and Nephritis" (3rd Edition) by Fishberg invaluable.

In concluding the question of etiology it seems wise to stress again two other correctable and often neglected factors in the production of the eclamptic state. These are obesity with uncontrolled weight gain in pregnancy, and focal infection. *A physician neglects his patient if he fails to note and to attempt to control her overweight at each prenatal visit quite as much as though he failed to observe her blood pressure and urine.* The matter of focal infection needs no further emphasis.

*Signs and Symptoms:* The well-known signs and symptoms of *relative* hypertension, albuminuria, edema, especially of the face, sudden weight gain, blurring of vision, nausea, and vomiting, constitute premonitory signals and sufficient cause whenever possible to hospitalize the patient. Torpor and irritability, mental or motor, constitute the final danger signals. Epigastric pain, convulsions, coma, and death within thirty-six to forty-eight hours too frequently constitute the termination of the eclamptic state. It is important to note that this condition progresses at different rates of speed, i.e., slowly in a matter of days or even weeks, rapidly in a matter of hours, or very rarely, fulminating—without warning. Since this is true, a weekly urine examination and a blood pressure and weight record every two weeks is the minimum requirement for proper prenatal care. Even then some "quick cases" will get off to a long start before detection. We have come to feel that borderline diastolic pressure, 90 to 100, is an important prognostic sign. If a patient shows any sign or symptoms and cannot be hospitalized for observation for economic reasons a daily urine specimen and a blood pressure reading every other day constitute the minimum requirement.

*Treatment:* Eclampsia, usually defined for simplicity as preeclampsia with the addition of convulsions, carries about ten times the maternal mortality rate of preeclampsia in this region. The *relative* fetal mortalities — if the child is over thirty-two weeks — is probably even greater in the two conditions. In 168 consecutive cases of eclampsia investigated recently we find a maternal mortality of nearly 40 per cent in emergency cases, nearly 20 per cent in observed cases, a combined mortality of just under 25 per cent. These cases occurred over a period of twenty years and were treated in a variety of ways, largely on conservative principles. No improvement in results can be demonstrated because of changed detail of treatment in these cases. *Therefore, since the maternal mortality in preeclampsia may be set at 2.5 per cent it seems reasonable to assert that to date the primary principle of the treatment of the "eclamptic state" is to empty the uterus, by the gentlest means and after getting the patient in her best possible condition by the well-known accepted sedative eliminative treatment, prior to convulsions.* This is usually satisfactorily accomplished by rupture of the membranes with or without the guarded use of a pressor-principle free oxytoxic. Occasionally abdominal cesarean section may be considered, but should be avoided if possible, since severe

preeclamptics are poor surgical risks. Nice judgment based on experience and a careful weighing of all factors including the preservation of the frequently premature baby are necessary to make this decision wisely, especially as it is a striking fact that a study of maternal mortality statistics in Massachusetts year after year invariably shows a notable list of combined "Toxemia — Cesarean Section — Peritonitis" deaths. It must be borne in mind that abdominal cesarean section in preeclampsia does not guarantee a living baby especially if it be premature. Clifford has shown a 45 per cent mortality in infants under five pounds in cesarean section done for this condition; a mortality of 27 per cent in the infants in this group that weighed from 4 to 5 pounds. Plass has shown recently a 6 per cent maternal mortality in sixty-seven sections for preeclampsia done throughout Iowa in 1930-1932. J. A. Smith of Boston reports a similar series of fifty-seven cases with a maternal mortality of 5.3 per cent. On the other hand the writer, paying especial attention to anesthesia with the omission of all preoperative medication and a technique of operation which exposes the baby to practically no anesthetic and the mother to very little has performed forty-five sections for severe preeclampsia with the death of a single mother, a rate of 2.2 per cent. Of the babies in this series twelve weighed between four and five pounds and were all discharged well from the hospital. Babies under four pounds, seven in number, were all less than twenty-eight weeks and all died. Babies over five pounds, twenty-five in number, all lived but one. The mother of this baby had preliminary medication including morphine forty-five minutes before operation. It may be added that four of the forty-five preeclamptics developed eclampsia. So perhaps it may be concluded that with an unfavorable cervix, abdominal cesarean section under special precautions has a place in the treatment of preeclamptic toxemia.

*Treatment of Eclampsia:* It is the generally accepted opinion that some one or other of the so-called conservative routine methods of treating the eclamptic state once the patient has developed convulsions (or coma) gives far better results than active obstetrical intervention. Further it is generally stated that cesarean section is, next to the long abandoned accouchement forcé, the method of treatment attended by the worst results. Yet one discovers that in most of the accepted routines some form of interference is permitted after so many hours or days of conservative treatment if improvement is not noted or the patient grows worse; and in studying many series of eclamptics treated conservatively on occasion the method of interference used appears to be cesarean section, so that we must conclude that it has a place, rarely at least, in the treatment of eclampsia also, after failure of conservative treatment. Latest figures, quoting Plass again, show that of eighty-four cesarean sections done for eclampsia in Iowa in 1930-1932, sixteen died a mortality rate of 19 per cent. If we compare this maternal mortality of eclampsia treated by cesarean section, all over one state, which implies in some instances at least a lack of detailed care, with the figures given earlier in this essay from an *individual* series treated in a variety of ways but practically all "conservative" we find that these cases treated by section give equally good results as the "under observation" group and twice as good results as the "emergency group". No fair conclusion can be drawn from these comparative statistics for a variety of reasons. The purpose of stressing them here is to emphasize that *method of treatment* and *method of delivery in eclampsia* are two, on the whole, separate propositions though they sometimes interlock. The question of *method of delivery* resolves itself into adding the least possible insult to an already heavily damaged body. From a practical point of view no delivery at all or normal delivery when possible does the least mischief. How much more eclampsia a given patient will take and live and how a given doctor views this aspect of the question is an individual matter and no routine can be laid down that meets the requirements of all cases. This is justifiable criticism of all "routine conservative treatment". Constant observation, treatment including rest, and individualized action depending on the results of these, will give the best results in eclampsia.

Among the untold number of remedies for and theoretical treatments of eclampsia, two deserve special mention. It has been shown that a relatively small proportion of eclamptic deaths result from cerebral hemorrhage, perhaps 10 per cent to 15 per cent. Such deaths are apt to occur in elderly women, probably with a hypertension background. The rest die of, or with, edema of the lungs or brain or both. The typical moribund eclamptic is clinically a cyanotic woman with wet lungs and running fluid from the mouth—the head lowered for drainage. Teel, Reid, and Hertig have shown that a few *preeclamptics* exhibit acute left ventricular failure, seemingly the result of the prolonged hypertension of the preeclampsia and not associated either with chronic nephritis or chronic heart disease; and in one instance death resulted in the attack. It seems likely that the wet lung moribund eclamptic is in the same state for the same reason. From these findings of cerebral edema and edema of the lungs it may be laid down as a sound principle that any treatment which may increase edema is unsound. Hence the theory of balanced fluids in the eclamptic state deserves special consideration. Since it is known that the damaged

kidney will put out only what fluids its condition allows, more fluids than this amount taken in must increase edema. If it is true on the above reasoning that increased edema is bad then it may be reasoned that any agent which tends to reduce edema, especially cerebral edema, probably the cause of the convulsions, is good. This brings us logically to the intravenous and intramuscular use of magnesium sulphate in eclampsia: a phase of the subject neglected in this locality.

Schwarz and Dorsett of St. Louis (*South. M. J.* **23:** 288, 1930) report 186 cases of eclampsia treated basically and conservatively with intramuscular or intravenous magnesium sulphate with a maternal mortality of 7 per cent. Rucker* of Richmond (1931) reported 109 personal cases of eclampsia treated with intravenous magnesium sulphate (20 cc. of a 10 per cent solution repeated p.r.n. for convulsions or impending convulsions) with a maternal mortality of 5.5 per cent. His previous mortality had ranged from 31.6 per cent under active obstetrical treatment to 25.9 per cent under other conservative treatment, i.e., morphine, venesection, gastric lavage, and colonic irrigation. He says of his 109 cases: "In only two cases was I unable to stop convulsions." And further to show that his mortality is not a corrected one, "Of the six deaths that occurred in the 109 cases of the ultraconservative group, two were not treated ultraconservatively, two died of infections after the eclampsia had subsided and only two were truly eclamptic deaths treated in the manner described" Lazard of Los Angeles and McCord of Atlanta report similar, good results by this treatment. Such results, especially Rucker's comparative ones in his own hands, are highly suggestive.

*In Conclusion:* We have indicated our belief in emptying the uterus prior to convulsions when possible in the eclamptic state. We have indicated that if the patient has passed the convulsion phase sharp individualization in *method of delivery* and *time of delivery* is necessary, and the conviction that, on necropsy and statistical grounds, an extended trial of intravenous or intramuscular magnesium sulphate treatment should be made hereabouts; and that balanced fluids constitute an additional important feature of treatment.

Each patient threatened with eclampsia should be hospitalized and given the benefit of obstetrical consultation.

*Rucker's last figures, 1934, show less than 5 per cent mortality in 123 cases. Digitalis for the prevention of pulmonary edema and venesection if it has occurred are added to the $MgSO_4$ treatment.

## AIDS TO THE COMMITTEE OF ARRANGEMENTS

### Essex South District Medical Society

Dr. Scott W. Mooring of Gloucester, Dr. Albert Parkhurst of Beverly, and Dr. Nathaniel Breed of Lynn, have recently been appointed from the Essex South District to assist the Committee of Arrangements for the Annual Meeting of the Massachusetts Medical Society at Springfield in June.

### Hampshire District Medical Society

Dr. Lawrence N. Durgin of Amherst, Dr. Thomas F. Corriden of Northampton and Dr. Stephen Brown of Northampton, have recently been appointed from the Hampshire District to assist the Committee of Arrangements for the Annual Meeting of the Massachusetts Medical Society at Springfield in June.

## FELLOWS OF THE MASSACHUSETTS MEDICAL SOCIETY!

Have you paid your annual dues?

Remember that if you are delinquent your *Journal* will not be mailed to you. If you wish to have your files intact, please attend to this obligation.

## MASSACHUSETTS LEGISLATIVE NOTES

**House Bill 59** which defines the words "stillborn child" has been enacted.

**House Bill 574** which was designed to provide that hospitals receiving public support should be required to accord equal rights of admission to patients of all duly registered physicians has been referred to the next annual session.

## MISCELLANY

### CHANGES IN SEVERAL BOARDS OF STATE INSTITUTIONS

According to current reports, His Excellency, Governor Curley, has nominated for the position formerly held by Frank B. Hall of Worcester, as Trustee of the Grafton State Hospital, Miss Martha Ducey of Shrewsbury.

Other nominations are the following: Monroe Kaplan of Boston to succeed Mrs. Esther M. Andrews of Brookline as a Trustee of the Boston Psychopathic Hospital; Michael McGrath of Salem to succeed Robert H. Sawyer of Haverhill; Mrs. Catherine Sullivan of Canton to succeed Mrs. G. S. Sutherland of Boston as Trustee of the Taunton State Hospital; Robert Portle of Worcester to succeed Howard W. Cowee of Worcester, Trustee of the Worcester State Hospital; John H. Craig of Natick to succeed Walter Channing of Dover as Trustee of the Medfield State Hospital; and William Bulman of Brockton to succeed Horace A. Keith of Brockton as Trustee of the Foxboro State Hospital.

Appointments as Trustees of the Massachusetts General Hospital: Miss Betty Dumaine, Henry V. Morgan, James H. Bushway, and Dr. Joseph Santosuosso.

Reappointments were Charles C. Cain of Taunton as Trustee of the Taunton State Hospital, and Charles F. Riordan of Sharon as Director of the State Division of Livestock Disease Control.

## AN ADDRESS BY DR. RUSHMORE

In response to an invitation by the Law Society of Massachusetts, Dr. Stephen Rushmore delivered an address before that body at the Boston City Club, February 25, 1936.

## A MEETING OF MEMBERS OF THE LEGISLATURE WITH PHYSICIANS

Worcester physicians entertained members of the Massachusetts Legislature from Worcester and vicinity Friday, February 21, at the Worcester City Club. The object of this meeting was the discussion of bills presented to the Legislature, and which have a bearing on medicine and especially on public health.

After enjoying a bountiful dinner the assembly was called to order by Dr. W. F. Lynch, President of the Worcester District Medical Society, who, after welcoming the guests, introduced Dr. A. W. Marsh of Worcester and a member of the Legislative Committee of the State Society, as master of ceremonies.

Dr. Marsh explained that this meeting was, according to precedent, an occasion for presenting to certain leaders in the State Legislature the opinion of Worcester physicians respecting those bills before the Legislature which bear on medical problems.

Several doctors, after being introduced by Dr. Marsh, submitted analyses of some of the more important bills, especially those relating to hospitals, various forms of medical practice and particularly House 34 introduced by the Board of Registration in Medicine.

Dr. Mongan, President of the State Medical Society, closed the discussion with a spirited appeal for the recognition of the importance of scientific medicine in dealing with the ills of the people and urged the protection of the citizenry against incompetent practitioners. In a supplemental admonition he emphasized the importance of an approved education for all physicians.

This meeting seemed to interest the members of the Legislature and is a commendable effort to secure coöperation.

## AFFAIRS IN CONNECTICUT

### Dr. C. C. Burlingame Speaks on "Withdrawing the Veil of Mystery that Surrounds the Mentally Ill."

Dr. C. Charles Burlingame, physician-in-chief of the Neuro-Psychiatric Institute and Hospital of the Hartford Retreat, addressed two lay audiences in Hartford, Conn., on January 31, 1936. His subject was treated in a most interesting manner. Condemning so-called mercy killing of the incurably diseased, Dr. Burlingame said:

"The helpless monstrosity might not have been deprived of anything worthwhile in a mercy killing, although even this is open to question. The sufferer from an incurable disease may have appeared to benefit by a mercy killing, although this is open to discussion. But one thing is certain: That man has not sufficiently sublimated his sadistic tendencies to permit mercy killings without grave dangers to society as a whole suffering from a release of man's imperfectly suppressed sadism."

The speaker pointed out that the first step in prevention of mental illness is a knowledge of the nature of the disease and its causes. He traced the etiology of some mental illnesses to the stresses and strains placed on the human mind by the civilizing process, in which certain destructive tendencies, natural in man, had to be curbed as a natural development of society. He cited war as an example of how instinctive destructive tendencies, barely covered by the thin veneer of civilization, can be brought suddenly to the surface.

"We need to think back no farther than our last war to see how quickly we brush aside the 'Thou shalt not kill' that we were brought up to regard as the greatest of the Ten Commandments, and under the pressure of military training, we have seen how easy it is to release sadistic tendencies and have millions of men accept with aplomb the job of killing.

"May I here interpolate that wars will not cease until there is a change in the brain of the human being. Peace pacts, leagues of nations, and balances of powers will not prevent the periodic release of man's sadistic tendencies until the individual man has developed much further. Wars will not disappear until the human brain makes a further development and finds a substitute for war."

Dr. Burlingame drew attention to the slight distinction which, he said, separates the normal from the abnormal, in the following illustrations:

"It is only a little step from a Dr. Samuel Johnson who felt obliged to touch every passing lamp post to the mentally ill person who must revolve seven times before sitting down."

"It is only a step from the irate golfer who smashes his club in anger and damns his club and the ball, to the mentally ill person who attributes a definite personality to the radio or to the chair in his room.

"It is only a step from the smut hunting censor who sees dirt in everything, to the mentally ill person who continually bathes himself to the exclusion of any productive activity in order that he may 'purify himself'.

"It is only a step from the ruthless dictator who wraps himself in a cloak of apostolic fervor and

thus makes his dream come true, to the hospital patient who in his manic drive develops delusions of personal grandeur to the point where he would readily undertake a reorganization of any portion of society with the assurance that the solution of all the difficulties of mankind lay in his hand."

Pointing out the prevalence of mental illness, Dr. Burlingame stated that a patient was admitted to a hospital for mental illness somewhere in the United States every four minutes of the day and night. At the regular rate of incidence, he said, out of approximately 338,000 children now in the grammar and high schools in Connecticut, 15,000 could be expected to be patients in hospitals for mental diseases some time during their lives.

Despite the magnitude of the problem, Dr. Burlingame expressed the belief that mental disease need not be the great calamity that many people regard it. He said it is no longer considered "the great destroyer we once thought it was" and "that along with other physical illness, it is in large measure preventable and fortunately curable to a very large degree." He decried the defeatist attitude of blaming everything on heredity as the cause of mental disease and said that other factors such as "mal-education and environment play an active part." Among a large group of mentally ill, he said, "Mental illness is merely a natural reaction to mal-education and faulty training of the emotions."

He pointed out that forms of mental illness due to glandular disturbances at certain periods in life are similar to mental disturbances which accompany purely physical disorders such as the delirium of typhoid fever, and that frequently, when the physical disease is cured, the mental symptoms disappear.

Dr. Burlingame emphasized the statement that almost every trait found in the mentally ill is an exaggeration of traits found in the community. "It is not difficult to understand the mentally ill person," he said. "He is as we are, only just a little more baffled by the world, just a little less able to adapt himself to it." He cited several historical cases of political and military leaders of the past, who, according to present-day knowledge, "had personalities which might be considered at least psychopathic".

The speaker advocated a more rational public attitude toward mental illness, with an increased knowledge of the necessity of preventing it by proper training, "spiritual education, emotional education, education of the man as a unit to the end that he is fitted to survive happily in his ever-changing environment".

He warned, in his conclusion, against an effort to protect children against the world, saying that too much protection was harmful.

"We must realize that the prolongation of the protected environment for the child and safeguarding him from the realities of life is part of his mis-education which may cause him to be among the 15,000 of today's children in Connecticut who will enter mental hospitals."

ST. FRANCIS HOSPITAL, HARTFORD, CONNECTICUT

At the annual meeting of St. Francis Hospital, Hartford, Conn., held on January 29, 1936, Dr. James F. Lynch was elected president of the Staff. Dr. John F. Dowling, president since 1916, resigned because of ill health. Dr. Lynch is a graduate of the College of Physicians and Surgeons, Baltimore, Md. Except for about one year while in the World War he has been connected with St. Francis Hospital since 1913.

Other staff promotions and new appointments are as follows: Dr. D. Dillon Reidy, chief of the urological service; Dr. Louis P. James, chief obstetrician; Dr. Henry Katz, from assistant to attending otolaryngologist and ophthalmologist; Dr. Joseph J. Connor and Dr. Edward A. Dignam, assistant otolaryngologists and ophthalmologists; Dr. Terence F. McNulty, assistant obstetrician; Dr. John T. Winters and Dr. Christopher J. McCormack, assistant surgeons; and Dr. R. W. Whitcomb, assistant oral surgeon.

## RECENT DEATHS

**CLAPP**—FRANK HORACE CLAPP, M.D., of North Grafton, Massachusetts, died at his home February 26, 1936. He was born in 1861. After graduating from the University of Vermont, he studied at the University of Vermont College of Medicine and graduated in 1888.

Before coming to Massachusetts, he practiced among the families of lumber camps, and later attended postgraduate clinics in New York City. Dr. Clapp was a fellow of the Massachusetts Medical Society, having joined in 1892, and also of the American Medical Association. He formerly served as President of the Worcester District Medical Society. He had served on the School Committee and the Board of Health of his town for several years and was active in many civic enterprises. Surviving Dr. Clapp are his widow, Mrs. Maud (Bailey) Clapp, a son, Dr. William B. Clapp of North Grafton, a daughter, Mrs. Florence Sanford, and a granddaughter, Barbara Sanford.

**CHASE**—AUGUSTUS LUCIUS CHASE, M.D., of Randolph, Massachusetts, died at his home February 29, 1936.

Dr. Chase was born in 1849 and graduated in medicine from the Eclectic Medical College, Cincinnati, in 1872. His prominence led to his appointment as one of the original members of the Board of Registration in Medicine in 1894, serving in this capacity continuously for twenty-seven years. For many years Dr. Chase served on the Brockton Pension Examining Board and was interested throughout his long life in various civic activities in Randolph.

Surviving Dr. Chase are two sons, Dr. Gilman L. Chase of Clinton, Massachusetts, Hon. Judge Herbert Chase of Cambridge, and a daughter, Mrs. Ella Cottle, of Bristol, Connecticut.

### DELAYED NOTICE*

**MASSÉ**—JOHN BAPTISTE MASSÉ, M.D., of 96 Bradford Street, Lawrence, Massachusetts, died suddenly October 15, 1935. Dr. Massé was born in Montreal, Canada, in 1876 and came to Lawrence with his parents early in life. He returned to Montreal to enter the University of Montreal Faculty of Medicine and graduated therefrom in 1902, and soon afterward opened an office in Lawrence for the practice of medicine. In this field he was very successful and popular.

He joined the Massachusetts Medical Society in 1907 and was a member of Lawrence Lodge 65, B. P. O. E.

Dr. Massé is survived by ten nieces and nephews all living in Canada except Emil Massé of Biddeford, Maine. Several cousins are residents of Lawrence.

*Recently received.

## NOTICES

### MEDICAL CLINIC AND STAFF ROUNDS AT THE PETER BENT BRIGHAM HOSPITAL

At 3:30 P.M. on Thursday, March 12, in the Amphitheatre of the Peter Bent Brigham Hospital, Dr. Samuel A. Levine, Senior Associate in Medicine, Peter Bent Brigham Hospital, will give a medical clinic. To it are cordially invited practitioners and medical students.

On Saturdays in the wards of the Peter Bent Brigham Hospital, from 10 to 12, staff rounds will be conducted.

### THE PHILADELPHIA COUNTY MEDICAL SOCIETY

PROGRAM FOR THE POSTGRADUATE INSTITUTE

Fifty-four of the prominent medical educators in a city noted for its medical education—Philadelphia—constitute the faculty of the Philadelphia County Medical Society's Postgraduate Institute, to be held April 20 to 24 in the Bellevue-Stratford Hotel, according to the complete program, just issued*.

Considerable interest has been expressed in this undertaking, which the county society hopes to make an annual event, and many physicians already have sent in their registrations. Notices have been sent to doctors of the nearby states and a large attendance is expected.

Dr. Frank H. Lahey will deliver the J. Chalmers DaCosta Foundation oration at the Philadelphia County Medical Society's meeting on the evening of April 22.

The Institute's general subject will be cardiovascular and renal diseases, which the essayists will discuss from many angles. One approach will be prevention, to which little attention was paid by doctors of the older schools. This is recognized as a very practical mode of attack today.

*A copy for examination is on file at the *Journal* Office.

### BOSTON UNIVERSITY SCHOOL OF MEDICINE SURGICAL CLINIC AT THE BOSTON CITY HOSPITAL

Friday, March 6, 12-1, Thorndike amphitheatre.

Dr. Frank H. Lahey will talk on "Thyroid Disease"; illustrated by lantern slides.

Physicians and medical students are invited.

### NOTICE

Reprints of the article by Dr. Francis T. Hunter under the title of Hutchinson-Boeck's Disease (Generalized "Sarcoidosis") will reproduce the illustrations more clearly than they appeared on pages 350 and 351 in the *Journal* of February 20.

### LAWRENCE CANCER CLINIC

Established April 17, 1928

Lawrence, Mass., March 2, 1936.

*To the Physicians of the North Half of Essex County:*

*Dear Doctor:*

The regular Lawrence Cancer Clinic, to be held at Lawrence General Hospital, One Garden Street, Lawrence, upon Tuesday, March 17, at 10:00 A.M., will be a Demonstration Clinic, with Channing C. Simmons, M.D., of Boston, Associate in Surgery in the Graduate Courses in Medicine at Harvard University Medical School, Surgeon-in-Chief to Collis P. Huntington Memorial Hospital, member of the Cancer Commission of Harvard University, Boston, and Visiting Surgeon to the Massachusetts General Hospital, present as consultant. You are invited to accompany any of your patients whom you desire shall have this service, or to send them with a note, and a report will be returned to you. The service is gratis. Your attendance at the Clinic is always welcome.

This clinic is endorsed by the Committee on Postgraduate Instruction of the Massachusetts Medical Society.

Committee:

ROY V. BAKETEL, M.D.,
CHAS. J. BURGESS, M.D.,
FRED'K D. MCALLISTER, M.D.,
JOHN J. MCARDLE, M.D.,
HARRY H. NEVERS, M.D.,
THOS. V. UNIAC, M.D.,
J. FORREST BURNHAM, M.D., *Chairman.*

## REPORTS AND NOTICES OF MEETINGS

### THE MASSACHUSETTS CENTRAL HEALTH COUNCIL

At the recent annual meeting of the Massachusetts Central Health Council the following designated officers were elected: Miss Sophie Nelson, R.N., of the John Hancock Mutual Life Insurance Company, President; Dr. Gaylord W. Anderson of the State Department of Public Health, Vice-President; and

Arthur J. Strawson of the Massachusetts Tuberculosis League, Secretary-Treasurer.

### WILLIAM OSLER HONORARY SOCIETY

A meeting of the William Osler Society was held February 13, 1936, at the Boston City Hospital. This is the honor medical society at the Tufts College Medical School, the membership of which is composed of highest ranking third and fourth year students. The meeting was arranged and conducted by graduate members, Carl M. Binnig, M.D.; Albert E. Sloane, M.D.; Harry H. Brenner, M.D., and William Fain, M.D.

From the class of 1936 the following students were elected to membership:

Joseph A. Reynolds, Sawyer Foster, Paul J. Catinella, Benjamin Stein, Max Stein, Morris Botvin, Stanley W. Machaj, Frank K. Duffy, Norman E. Peatfield, Mildred Adell, Jacob Mezer, Max Goldman and Israel Zeltzerman.

Formal induction of the new members will take place at the Hotel Lenox, April 14, 1936.

### THE ARLINGTON DOCTORS' CLUB

The regular meeting of the Arlington Doctors' Club will be held at the Nurses' Home of the Symmes Arlington Hospital on Tuesday evening, March 10, 1936, at 8:30 P.M.

The speaker will be Dr. Frank H. Lahey, director of the Lahey Clinic. His subject will be "Diseases of the Thyroid and Parathyroids."

The talk will be illustrated with lantern slides.

There will be a general discussion.

The members of the Somerville Doctors' Club have been invited to attend.

All physicians are welcome.

FRANK H. GERRY, M.D., *President,*
SIDNEY M. SIMMONS, M.D., *Secretary.*

### WILLIAM HARVEY SOCIETY

The next meeting of the William Harvey Society will be held Friday, March 13, in the Auditorium of the Beth Israel Hospital, Boston, at 8:00 P.M.

PROGRAM

Speaker: Dr. Alexander Lambert, formerly Professor of Clinical Medicine, Cornell University Medical School.

Subject: Therapeutics of Drug Habits.

Chairman: Dr. Harry Linenthal, Clinical Professor of Medicine, Tufts College Medical School.

### SOUTH END MEDICAL CLUB

The next regular meeting of the South End Medical Club will be held at the office of the Boston Tuberculosis Association, 554 Columbus Avenue, Boston, on Tuesday, March 17, 1936, at 12 noon. The speaker will be Frank H. Lahey, M.D., Harvard University Medical School 1904; F.A.C.S.; Director, Lahey Clinic; Surgeon-in-Chief, New England Baptist Hospital; Surgeon, New England Deaconess Hospital. His subject will be "Influence of Thyroid Disease in General Practice." All physicians are cordially invited to attend. Luncheon will be served.

### AN ADDRESS BY DR. KENDALL EMERSON

On April 8 at Hotel Kimball, Springfield, Dr. Kendall Emerson, Managing Director of the National Tuberculosis Association, New York, will address a joint annual meeting of the Massachusetts Tuberculosis League and the Hampden County Tuberculosis and Health Association. A program in the afternoon and the two board meetings will precede the dinner meeting.

### GREATER BOSTON BIKUR CHOLIM HOSPITAL

Greater Boston Bikur Cholim Hospital medical meeting Wednesday evening, March 18, at 8:30 o'clock, at the Nurses' Home, 45 Townsend Street, Roxbury. Speaker: Dr. Herrman L. Blumgart. Subject: Treatment of Angina Pectoris. The profession is invited.

HENRY BAKER, M.D., *Secretary.*

### HARVARD MEDICAL SOCIETY

The next meeting of the Harvard Medical Society will be held in the Peter Bent Brigham Hospital Amphitheatre (Shattuck Street Entrance), Tuesday evening, March 10, at 8:15 P.M.

PROGRAM

Presentation of Cases.

Down the Lymphatics with Camera and Cannula. By Dr. John Homans.

Medical students and physicians are cordially invited to attend.

MARSHALL N. FULTON, M.D., *Secretary.*

## SOCIETY MEETINGS, CONGRESSES AND CONFERENCES

### CALENDAR OF BOSTON DISTRICT FOR THE WEEK BEGINNING MONDAY, MARCH 9, 1936

**Tuesday, March 10—**

*9-10 A.M. Boston Dispensary, 25 Bennet Street, Boston. Mistakes Made in the Diagnosis and Treatment of Syphilis. (Continued.) Dr. F. M. Thurmon.

2:30 P.M. Pediatric Ward Visit. Massachusetts Eye and Ear Infirmary.

*8:15 P.M. Harvard Medical Society. Peter Bent Brigham Hospital Amphitheatre (Shattuck Street Entrance).

*8:30 P.M. The Arlington Doctors' Club. Nurses Home, Symmes Arlington Hospital.

**Wednesday, March 11—**

*9-10 A.M. Boston Dispensary, 25 Bennet Street, Boston. Indications for Radiation Therapy. Dr. C. E. Dumas.

†12 M. Clinico-Pathological Conference. Children's Hospital.

**Thursday, March 12—**

*8:30-9:30 A.M. Clinic, Surgical and Orthopedic Staffs of Children's Hospital, at the Children's Hospital.

*9-10 A.M. Boston Dispensary, 25 Bennet Street, Boston. Gastrointestinal Clinic. Dr. K. S. Andrews.

*3:30 P.M. Medical Clinic at the Peter Bent Brigham Hospital.

**Friday, March 13—**

*9-10 A.M. Boston Dispensary, 25 Bennet Street, Boston. Lung Abscess. Dr. Frederick T. Lord.

12 M. Massachusetts General Hospital. Clinical Meeting of the Staff of the Children's Medical Service. Ether Dome.

8 P.M. William Harvey Society. Beth Israel Hospital, Boston.

**Saturday, March 14—**

*9-10 A.M. Boston Dispensary, 25 Bennet Street, Boston. Hospital Case Presentation. Dr. S. J. Thannhauser.

*10-12. Staff rounds at the Peter Bent Brigham Hospital.

**Sunday, March 15—**

4 P.M. Free Public Lecture, Harvard Medical School, Building D, Longwood Avenue. Hearing and Its Conservation. Dr. Hallowell Davis.

*Open to the medical profession
†Open to Fellows of the Massachusetts Medical Society.

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**March 5**—Faulkner Hospital Clinical Meeting at 5 P.M.

**March 6**—Boston University School of Medicine Surgical Clinic at the Boston City Hospital. See page 497.

**March 6**—American Society for the Control of Cancer. See page 398, issue of February 20.

**March 10**—Harvard Medical Society. See page 498.

**March 10**—The Arlington Doctors' Club. See page 498.

**March 12**—Medical Clinic, Peter Bent Brigham Hospital. See page 497.

**March 13**—William Harvey Society. See page 498.

**March 17**—South End Medical Club. See page 498.

**March 17**—Lawrence Cancer Clinic. See page 497.

**March 18**—Greater Boston Bikur Cholim Hospital. See page 498.

**March 30**—Springfield Medical Association, 8:30 P.M. at the rooms of the Springfield Academy of Medicine, 20 Maple Street. The Development of Surgical Practice in Springfield. Dr. John M. Birnie.

**April 8**—Joint Meeting of the Massachusetts Tuberculosis League and the Hampden County Tuberculosis and Health Association. See "An address by Dr. Kendall Emerson." Page 498.

**April 20-24**—A Postgraduate Institute in Philadelphia. See page 497.

**May 12-16**—The International Congress of Physical Medicine. See page 443, issue of February 27.

**June 15-19**—The Executive Board of the Catholic Hospital Association will meet at the Fifth Regiment Armory, Baltimore, Md.

**June 16-July 28**—Summer Course in Bacteriology. See page 385, issue of February 20.

**September, 1936**—First International Conference on Fever Therapy. See page 1325, issue of December 26, 1935.

**October 19-23**—Clinical Congress of the American College of Surgeons. See page 180, issue of January 23.

## DISTRICT MEDICAL SOCIETIES

### ESSEX SOUTH DISTRICT MEDICAL SOCIETY

**April 1—Wednesday. Essex Sanatorium, Middleton. Clinic 5 P.M. Dinner 7 P.M. Speaker: Dr. Richard H. Overholt of the Lahey Clinic. Subject: Chest Surgery.**

**May 7—Thursday. Censors' Meeting.**

**May 13—Wednesday. Annual Meeting. Salem Country Club. Dinner at 7 P.M. Speaker: Dr. Paul White. Subject to be announced later.**

R. E. STONE, M.D., Secretary.
88 Lothrop Boulevard, Beverly.

### FRANKLIN DISTRICT MEDICAL SOCIETY

Meetings are held on the second Tuesdays of March and May at the Weldon Hotel, Greenfield, at 11 A.M.

CHARLES MOLINE, M.D., Secretary.
Sunderland.

### MIDDLESEX EAST DISTRICT MEDICAL SOCIETY

Meetings to be held at the Bear Hill Golf Club, Stoneham, at 12:15 P.M.

**March 11, May 6.**

K. L. MACLACHLAN, M.D., Secretary.
1 Bellevue Avenue, Melrose.

### NORFOLK DISTRICT MEDICAL SOCIETY

**March 31**—Hotel Kenmore, at 8 P.M. Dr. Benedict F. Boland—Cauterization of the Cervix Uteri Using Various Electrical Methods. Illustrated with lantern slides.

**May**—Annual Meeting. (Place, date and subject to be announced.)

The censors meet for the examination of candidates: May 7, 1936; November 5, 1936.

FRANK S. CRUICKSHANK, M.D., Secretary.
1236 Beacon Street, Brookline.

### PLYMOUTH DISTRICT MEDICAL SOCIETY

**March 19**—Plymouth County Sanatorium, South Hanson.

**April 16**—Brockton Hospital.

**May 21**—Lakeville State Sanatorium.

G. A. MOORE, M.D., Secretary.
167 Newbury Street, Brockton.

### SUFFOLK DISTRICT MEDICAL SOCIETY

**March 18—Meeting at the Boston Medical Library. The Laboratory and Clinical Story of Fatigue, Dr. Arlie V. Bock and Dr. David B. Dill. Discussion: Dr. Donald J. MacPherson and Dr. Augustus Thorndike, Jr.**

**April 29—Annual Meeting at the Boston Medical Library. The Treatment of Septicaemia, Dr. Champ Lyons. The Pleurality of Scarlatinal Streptococcus Toxin, Dr. Sanford B. Hooker. Discussion: Dr. Hans Zinsser.**

**The medical profession is cordially invited to attend these meetings.**

ROBERT L. DeNORMANDIE, M.D., President,
CHARLES C. LUND, M.D., Secretary.

### WORCESTER DISTRICT MEDICAL SOCIETY

**March 11—Wednesday evening. Memorial Hospital, Worcester, Mass. Dinner and scientific program.**

**April 8—Wednesday evening. Hahnemann Hospital, Worcester, Mass. Dinner and scientific program. Subjects of program to be announced later.**

**May 13—Wednesday afternoon and evening. Annual Meeting of Society. Time, place and details of program to be announced in an April issue of the Journal.**

ERWIN C. MILLER, M.D., Secretary.
27 Elm Street, Worcester.

## BOOKS RECEIVED FOR REVIEW

**The Diagnosis and Treatment of Pulmonary Tuberculosis.** A handbook for practitioners; a textbook for students, nurses and social workers. John B. Hawes, 2nd, and Moses J. Stone. 215 pp. Philadelphia: Lea & Febiger. $2.75.

**Reports on Chronic Rheumatic Diseases.** Annual Report of the British Committee on Chronic Rheumatic Diseases. Number One. Edited by C. W. Buckley. 159 pp. New York: The Macmillan Company. $4.00.

**Doctor of the North Country.** Earl Vinton McComb. 238 pp. New York: Thomas Y. Crowell Company. $2.00.

**The Art of Ministering to the Sick.** Richard C. Cabot and Russell L. Dicks. 384 pp. New York: The Macmillan Company. $3.00.

**Annual Report of the Surgeon General of the Public Health Service of the United States for the Fiscal Year 1935.** 158 pp. Washington: United States Government Printing Office. 75c.

**Clinical Miscellany. The Mary Imogene Bassett Hospital, Cooperstown, New York.** Francis F. Harrison, Charles C. McCoy, et al. Volume II: 1935. 218 pp. Springfield and Baltimore: Charles C Thomas. $3.00.

**The Diagnosis and Treatment of Diseases of the**

**Peripheral Arteries.** Saul S. Samuels. 260 pp. New York: Oxford University Press. $3.50.

**Le Thymus: Anatomie — Histologie—Physiologie Clinique et Thérapeutique.** G. Worms and H. Pierre Klotz. 152 pp. Paris: Masson et Cie. 30 fr.

**Endocrinologie.** Noël Fiessinger. 152 pp. Paris: Masson et Cie. 20 fr.

**You Must Eat Meat.** Fancies, Foibles and Facts about Meat. Max Ernest Jutte. 164 pp. New York: G. P. Putnam's Sons. $2.00.

**Lobar Pneumonia and Serum Therapy: With Special Reference to the Massachusetts Pneumonia Study.** Frederick T. Lord and Roderick Heffron. 91 pp. New York: The Commonwealth Fund. $1.00.

**A Manual of the Common Contagious Diseases.** Philip Moen Stimson. 437 pp. Second Edition, Thoroughly Revised. Philadelphia: Lea & Febiger. $4.00.

## BOOK REVIEWS

**An Introduction to Public Health.** Harry S. Mustard. 250 pp. New York: The Macmillan Company. $2.50.

This book is the fruit, both of practical experience with the actual problems involved and of teaching public health to students, graduate and undergraduate, and to nurses. In it the general principles of preventive medicine, as they may be practically applied by society to the prevention of disease, are treated in perspicuous fashion and with a nice regard for relative values. The style is pungent and lucid, although the author may be mildly criticized for the use of too many "et ceteras." The subjects treated are as follows: The background of public health, vital statistics, organization and administration of public health work, the acute communicable disease, tuberculosis as a public health problem, the venereal diseases, sanitation, personal hygiene, the hygiene of infancy and childhood, school health service and noncommunicable diseases. This is not only an excellent textbook for the student, but because of its clear exposition, could be read and studied to great advantage by the intelligent citizen.

**Complete Handbook on State Medicine.** J. Weston Walch. 158 pp. Portland: Debaters Information Bureau. $2.50.

This is a compilation of articles and opinions relating to problems incident to medical care for the people of the United States and is especially designed for study by those who may use the material in debates.

The essential arguments pro and con relating to the costs of medical care and the suggestions of interested individuals and groups as to methods designed to meet the needs of the people are set forth in this publication. Explanations of the various forms of "health insurance", state, socialized, public and industrial medicine are set forth.

Although this book is designed to make available information relating to the economic problems of dealing with illness, there is a great deal of information between the covers which will interest economists, sociologists, and physicians.

Groups interested in carrying on debates relating to these questions will find useful material in the book.

**John Whitridge Williams. Academic Aspects and Bibliography.** J. Morris Slemons. 109 pp. Baltimore: The Johns Hopkins Press. $1.50.

Dr. Slemons has given us a delightful account of the life and accomplishments of an outstanding man in American medicine. An investigator, a teacher, a writer, Dr. Williams stands preëminent among the American physicians of our time, and we are grateful to Dr. Slemons for giving us such a stimulating account of his life.

**The Theory and Practice of Anaesthesia.** M. D. Nosworthy. 223 pp. London: Hutchinson Scientific. 12/6 net.

It is a pleasure to find a textbook of anesthesia that is modern in point of view and up-to-date in subject matter. Such recent additions to the field as cyclopropane, divinyl ether (vinethene), nembutal, evipal, and the carbon dioxide absorption method are well, though briefly, discussed. The chapters on premedication, nitrous oxide, carbon dioxide, acidosis, and difficulties in general anesthesia are of outstanding worth. Other subjects that are well treated are ether, chloroform, the endotracheal method, the mode of action of general anesthetics, and their aftereffects. Ethyl chloride, shock, and the stages and signs of general anesthesia are covered satisfactorily. The chapter on choice of anesthetics unfortunately rather slights spinal anesthesia as well as the newer and less frequently used drugs and methods. The section on spinal anesthesia is a distinct disappointment in that it advises neglect of blood pressure, and does not deal with the two drugs that are most commonly used in this country, namely, procain (novocain, neocain) and pontocain (pantocain). The book lacks both a chapter on regional anesthesia and a description of any gas machine commonly used in this country.

The author shows a wide and thorough knowledge of the field, and in place of the prejudices so often evident in writings on medical specialties, exhibits well-balanced judgment. The book is brief, the style direct, and the pages are filled with innumerable minor helpful bits of advice. It is the only book on general anesthesia known to the reviewer that he can warmly recommend as an up-to-date, brief, practical course of instruction for medical students as well as for those physicians who only occasionally administer anesthetics. For those who wish to specialize in anesthesia it is recommended as giving a good foundation in practical work which would serve as a starting point for further studies.